THE PREVALENCE OF RHEUMATIC DISEASE IN A RURAL COLOURED POPULATION IN NAMAQUALAND

By

ORLANDO LLEWELLYN MEYERS MBChB FCP (SA)

A thesis presented for the degree of

Doctor of Medicine

University of Cape Town

July 1982

For Meg and Paul and Clare

ACKNOWLEDGEMENTS

No man is an island entire of itself
every man is a piece of the Continent,
a part of the main.

Devotions XVII. J.Donne. 1571-1631.

ACKNOWLEDGEMENTS

One is part of a whole, and the role one fulfils is really the result of the endeavours of many others, who make it possible to live an enjoyable life and make it meaningful. This work will not have been possible were it not for willing and unstinted assistance of many individuals, and institutions. I wish to place on record my indebtedness to the following:

Bishop John Minder, Bishop of Keimoes in whose diocese Rietpoort falls, who always opened doors.

Father van den Berg and Father Chevalier and the Oblate Sisters of the Rietpoort Mission particularly Sister Marie Hildegard, for their tremendous support and for their example of service.

The Management Board of Rietpoort for making the process of this field work easier.

The people of Rietpoort-Putsekloof, Molsvlei-Stofkraal, and Lepelsfontein, whose enthusiasm for the project was a spur to all of us.

Dr. Sue Jessop who provided the major support in the preparation and conduct of the field work.

Dr. Pat Klemp, Dr. Marcus Fredman and Dr. Nick King who provided help in several areas of the field work.

Sister Meg Meyers, Avril Downie and Ethne Woolf who carried the burden of ensuring that the questionnaires were filled in and who took charge of the onerus task of labelling specimen tubes, taking blood etc.

Angela Langman and Julie Summers, the radiographers, for the radiography and without whose 'mechanical' endeavours this essential part of the survey would have failed.

Mr. Arthur Houston, my father-in-law, for the general help and for conveying specimens to the rail head at Bitterfontein, and for his constant encouragement to complete this thesis.

Miss Magriet Owies who acted as the registration clerk.

Mr. Edgar Carelse who provided the laboratory technical support.

The Medical Research Council, the University Staff Research and the Isaac Albow Rheumatology Bequest for the financial support and the Arthritis Foundation for the use of their combi. In particular the Medical Research Council provided the mobile unit, and its able driver Mr. Attwell Stamper.

The Medical Superintendent, Dr. H. Reeve-Sanders who permitted the use of hospital screens.

Professor S. Benatar for the provision of vitalographs and other equipment.

Dr. M. Keraan and Mrs. G. Cridland who provided the home based laboratory knowledge and for their careful and meticulous laboratory work.

The Namaqualand Bus Service who kindly conveyed the specimens to Cape Town free of charge.

My Secretary, Mrs. Pearl Symons for typing the rough drafts and the final version of this thesis.

Miss.S. Katcher and the Staff of the Medical Library, and the Staff of the Archives for generous help and the use of their facilities.

Professor. M.C. Berman, who provided the Staff and the facilities for measuring the serum uric acid and the serum iron.

Finally I have to express my deep appreciation and love to my wife, Meg, whose unselfish love and unfailing support, and my children, Paul and Clare, for their quiet support and understanding, while the thesis was in writing.

PREFACE

PREFACE

No epidemiological field studies of the rheumatic diseases have been undertaken in the Coloured population of South Africa. As a group they are genetically heterogenous and in comparative medicine they have tended to occupy an intermediate position between Black and White South Africans. A study of the nature and extent of rheumatic disease in this population group can make an important contribution.

(i) The Coloured population is both simple, unsophisticated and rural, and on the other hand unquestionably urbanised. In this sense they provide a unique opportunity to study the prevalence of rheumatoid arthritis under rural and urban conditions. Such a study if it supports the rural/urban differences which have been shown for Black South Africans will help to focus attention on the urban environment in the pathogenesis of rheumatoid arthritis. The same may be true of other rheumatic diseases such as systemic lupus erythematosus. On the other hand data which is similar to that of other studies also has importance in reinforcing accepted ideas, and furthermore such a study affords an opportunity to test criteria of disease in differen. circumstances. This helps to define problems and ultimately aids in the refining of criteria.

(ii) The planning of strategies for health services cannot hope to become adequate neither can the effectiveness of such health strategies be measured if the prevalence of an important group of diseases is not known.

(iii) The teaching of under and postgraduates must also be influenced by the research conducted by a medical school, and in this way the provision and the planning of health services are aided.

This study in a rural Coloured population forms the first of a series of studies in rural/urban living Coloured people. The rural study used as its universe the population of Rietpoort in Namaqualand, where 80% of the adult population was seen.

INDEX

INDEX

	Page
CHAPTER 1 INTRODUCTION	1 - 28
The epidemiology of rheumatic disease	1
The rheumatic diseases in South Africa	5
CHAPTER 2 A BRIEF GLIMPSE OF HISTORY	
The geography of Rietpoort	29
The origin of the Coloured people	31
The establishment of Rietpoort	31
The role of the Church at Rietpoort	34
The reasons for choosing Rietpoort for the study	35
CHAPTER 3 METHODOLOGY	36 - 47
The census and the plan of the field work	36
The evaluation of rheumatic disease	
(i) Historical recall of joint disease	36
(ii) The clinical examination	37
Laboratory Tests	
(i) Haemoglobin, white cell counts	39
(ii) Serum complement	40
(iii) Rheumatoid factor	41
(iv) Auto-antibodies	41
(v) Serological tests for syphilis	43
(vi) Serum uric acid	43
(vii) Serum electrophoresis/serum syphilis	43

Radiological examinations

(i) Erosive arthritis 44

(ii) Osteoporosis 44

(iii) Osteoarthrosis 45

(iv) Hallux Valgus 46

CHAPTER 4 DEMOGRAPHY 48 - 54

1) The census of Rietpoort 48

2) Completion rates 49

3) Residential features/occupation/income 50

4) Anthropomorphic measurements 52

5) Serum albumin levels 53

CHAPTER 5 PREVIOUS HISTORY OF ARTHRITIS AND/OR BACKACHE 55 - 64

Previous history of arthritis 55

Lack of residua 57

Causes of 'Benign' Polyarthritis 58

Previous history of backache 60

The symptom profile of backache 62

The epidemiology of backache 63

CHAPTER 6 RHEUMATOID ARTHRITIS 65 - 89

Pathogenetic mechanisms 65

The Criteria for Rheumatoid arthritis 69

The application of the Criteria in Rietpoort 73

Comparison with other South African studies 74

Comparison with other studies (NY Criteria) 75

Prevalence of Rheumatoid arthritis in rural/urban areas 76

The prevalence of Rheumatoid arthritis in Africa 79

Is Rheumatoid arthritis a new disease? 80

Erosive arthritis 81

Juvenile chronic arthritis 83

Psoriasis/psoriatic arthropathy 84

Ankylosing spondylitis 85

Crystal arthritis 86

Systemic Lupus erythematosus 87

Unexplained arthritis 87

CHAPTER 7 OSTEOARTHROSIS 90 - 105

Semantic problems 90

Pathology of osteoarthrosis 90

Diagnosis and evaluation of osteoarthrosis 94

The prevalence of osteoarthrosis at Rietpoort 95

Metacarpophalangeal osteoarthrosis 103

CHAPTER 8 SOFT TISSUE RHEUMATIC DISORDERS 106 - 127

Shoulder, the clinical evaluation of 106

Shoulder pain in the Rietpoort population 108

The normal range of movement at the shoulder 109

The prevalence of shoulder disease in Rietpoort 111

Lesions of the rotator cuff 112

Adhesive capsulitis 115

Unexplained shoulder problems 117

Previous shoulder trauma 119

Lateral epicondylitis 120

The causes of lateral epicondylitis 122

Bursitis 123

Dupuytren's contracture 124

Flexor tendon disease of the fingers 124

De Quervains tenosynovitis 125

The carpal tunnel syndrome 125

The Fibromyalgic syndrome 126

CHAPTER 9 OSTEOPOROSIS 128 - 139

Osteoporosis, semantic problems of 128

The physiology of bone 128

The classification of osteoporosis 130

The importance of osteoporosis 131

Methods of evaluating osteoporosis 131

Method used in the Rietpoort population 133

The age specific prevalence of osteoporosis 135

CHAPTER 10 CAMPTODACTYLY, CLINODACTYLY, BONE AND JOINT TRAUMA, HALLUX VALGUS AND ARTICULAR HYPER-MOBILITY 140 - 152

Clinodactyly 140

Camptodactyly 140

Bone/Joint Trauma 141

Hallux valgus 142

Articular hypermobility 150

CHAPTER 11 AUTO-ANTIBODIES, SERUM COMPLEMENT AND SERUM URIC ACID 153 - 176

Anti-nuclear Factor 153

Extractable nuclear antigen 155

Smooth muscle antibody 158

Rheumatoid factor 160

Anti thyroid auto-antibodies 162

Comparison with other populations 163

Total haemolytic complement 165

Serum uric acid 165

Serological tests for syphilis 170

CHAPTER 12 ILLNESS PROFILE 171 - 176

Analysis of the Hospital register 171

Medical problems seen during the survey 173

Hypertension in Rietpoort 174

CONCLUSION 177 - 179

BIBLIOGRAPHY 180 - 235

A LIST OF ILLUSTRATIONS (between page 54/55)

The Rietpoort Mission X

The environs of the Mission X

Some of the children of Rietpoort XI

An elderly member of Rietpoort XI

Water is channelled from the granite outcrops into storage tanks XII

Collecting household water XII

The houses occur in small clusters XIII

The homes are simple with none of the amenities of a town/city XIII

Starting the day XIV

The M.R.C. Mobile X-Ray/Laboratory XIV

Waiting for the X-Ray XV

The Arthritis Foundation Combi was used to fetch the elderly and infirm XV

ILLUSTRATIONS OF RADIOGRAPHS (following page 105)

Osteoarthrosis of the metacarpophalangeal joints XVI

APPENDIX XVII - XXVIII

CHAPTER 1

I shall ever regret that I passed through such a country as the Haardeveldt and the confused granite masses of the Kamiesberg and Concordia almost blindfold, and only began to open my eyes as I was leaving Namaqualand.

Charles D.Bell. 1855.
Reports of the Surveyor General on the Copperfields of little Namaqualand.

Gilbert Ryle once described epidemiology as the pathology of families, societies or large populations. The discipline sets out to study the distribution and determinants of disease frequency in man. The distribution describes health status in terms of age, sex, race, geography and this aspect is a logical extension and application of the discipline of demography. The determinants of disease are concerned with the description of disease in causal terms. All the subdisciplines of medicine seek to learn about the determinants of disease, but epidemiological method provides a special contribution from the use of the knowledge about the frequency and distribution of disease in a population (MacMahon and Pugh 1970). As a discipline epidemiology depends heavily upon medicine and pathology for the descriptions of disease to give it direction and validity and upon biostatistics for the numerical handling of the data. From having been concerned primarily with infective disease and epidemics the epidemiologist now stretches the concept of epidemics to include concepts of excessive or reduced prevalence of disease, and increasingly with the application of epidemiological knowledge to the equitable distribution of resources and the planning of health strategies (Allander and Bjelle 1981).

Epidemiology can thus be resolved into three dominant activities:

(1) Counting the frequency of occurrence of disease.

(2) Comparing the frequency of occurrence of a disease under two or more different circumstances.

(3) Drawing inferences and making judgements about the occurrence of disease.

As a discipline it has acquired an esoteric ambience much of which arises from its concern with sophisticated statistical procedures, and the perceived, infrequent and indirect impingement on individual patient care. It is true that it represents a radical departure from individual orientated medical practice, but the two are not incompatible, rather complementary. Epidemiology should be regarded as the 'afferent' limb of health service planning, because it provides the only means by which the magnitude of various diseases can be expressed and som appreciation of their relative importance can be judged (Roberts 1977).

The rheumatic diseases currently comprise 180 nosological entities, many of which are difficult to diagnose because of the absence of pathognomonic signs or definitive laboratory tests. A consequence of this is that diagnosis in rheumatology is critically dependant on; (i) the care with which the historical account of the illness is handled; (ii) a careful clinical examination and the application of history and clinical features to form criteria for diagnosis and a classification. One of the difficulties which has bedevilled the epidemiology of the rheumatic diseases has been the lack of uniformity of acceptable criteria for use in field work. Under ideal circumstances such a definition of disease should clearly separate those affected from those unaffected, but herein lies a problem with many of the rheumatic diseases; there are few pathognomonic signs and as a result the criteria are not always sufficiently sensitive nor specific. Rheumatoid arthritis poses particular dilemmas for those who are intent on measuring its

frequency in a population, because no specific definition exists. For instance is it an inflammatory polyarthritis (embracing all forms of nonspecific joint disease), and is the most reliable criteria of the inflammatory joint disease an erosive arthritis or should we accept the disease as an immunopathological one and take rheumatoid factor as its hallmark. The problem would seem to be pedantic if we are to base our views on the type of disease which is generally seen in hospitals. There the more chronic forms appear to resolve themselves into fairly easily recognisable entities, but in populations a milder form predominates and it appears more homogenous. The difficulties with criteria have been well described (Masi 1967) for the ARA criteria where specificity and sensitivity were evaluated. Three of the criteria would be 88% sensitive and 77% specific, compared with 5 or more criteria which would be 70% sensitive and 91% specific. Although this may seem to hold out hope for epidemiologists such a specificity of 91% is synonymous with a 9% false positivity. This is clearly a problem with a disease with a low prevalence such as rheumatoid arthritis. Despite the limitations, many able minds have contributed to criteria which, although imperfect, nevertheless do have utility and do give reasonably acceptable predictions of disease prevalence. Tribute must be paid to these people because they were some of the first to exploit the epidemiological approach in chronic disease, and they were among the first to recognise the need for standardisation (Wood 1969).

An examination of the economic and personal cost of rheumatic disease shows that the effects of rheumatic illness are enormous. In Britain it is estimated that during the course of a year more than 8 million people will consult a general practitioner for some sort of rheumatic complaint, and that 23% of patients seen by general practitioners have some sort of rheumatic disease.

From an economic point of view the loss of productivity from arthritis/rheumatism during 1972 - 1973 was £420 million (of this 25% was for back pain alone!), and an estimate of wages lost is of the order of £260, and the replacement of homemakers is estimated at £50 - 100 million. During 1972/1973 the State paid out £85 million in social security benefits for rheumatic disease (Wood 1977; Nuki, Brookes, Buchanan 1972).

The personal cost of rheumatic disease is also considerable. In Britain estimates are that more than a million people are impaired by rheumatic disorders and at least 20% of these are severely disabled by arthritis. Some measures of disadvantage include the following; 3% are confined to a bed/wheelchair, 12% are unable to leave their homes, 17% are unable to bath themselves and 20% have had to give up work, and 2% had to change their occupation (Wood 1977). The time spent with rheumatic patients is also greater than the average calculated for office based physicians (Epstein and Henke 1981; Gaffney 1979). Despite these rather gloomy figures the scope and the challenge

of the rheumatic diseases can be paraphased as follows:
'Most doctors whatever their speciality still deal with diseases which have to be lived with (by the patient, the doctor as well as the family, the employer, the doctors secretary and a host of paramedical personnel) and furthermore lived in the home. Other aspects and needs of society in general e.g. research teaching, economy in drug prescribing, genetic counselling, epidemological aspects of disease and guidance of the determinants of social priorities are all encompassed in the scope 'and the challenge' of rheumatology' (Bywaters 1973).

RHEUMATIC DISEASE IN SOUTHERN AFRICA

The rheumatic diseases have not received much attention in Southern Africa. This is in part because of the demands which nutritional and infective diseases have made on the medical services, and in part also because of uniformly prevalent views that these diseases are uncommon and untreatable. The nature of rheumatic disease which occurs in South Africa is not known with certainty, but there are a few studies and reports which give some idea about them. In one study of the pattern of illness seen by fifteen general practitioners in Cape Town, the rheumatic diseases contributed approximately 10% to the illness profile (Silbert 1970). The types of rheumatic disease were very little different from those seen in a rheumatological practice in America (Bohan 1981) although more serious disease tends to be emphasised in a private rheumatological practice. In a similar attempt to define the illness profile of Caucasians attending a general practice in Salisbury the following information

was given, rheumatoid arthritis 5, Collagen disease 5, osteoarthritis 29 and musculoskeletal disease 240. These diagnoses were recorded in 3500 patients (Castle and Bernberg 1969). This information is interesting because the rheumatic diseases mentioned account for 8% of all diseases encountered. This figure is remarkably similar to that of the Cape Town Illness Profile. A study of the causes of chronic disability in non-institutionalised persons in the metropolitan area of Cape Town contains further information about rheumatic disablement. This study has shown that diseases of the musculo-skeletal system rank 1st - 3rd as a cause for chronic disability in White, Coloured and Blacks (Dick, Spencer, Watermeyer 1978). Another way of trying to form an estimate of the prevalence and the cost of rheumatic disease can be computed from data of the National Drug and Therapeutic Index for 1979 and 1980 (Table 1.1.)

TABLE 1.1.

PRESCRIPTIONS FOR NON-STEROIDAL ANTI-INFLAMMATORY DRUGS (NSAI) IN SOUTH AFRICA

	1979	1980
Total prescription for NSAI drugs	2,912,000	3,414,000
Unspecified rheumatic disease	313,000	373,000
Non-articular rheumatism	388,000	435,000
Backache	35,000	44,000
Osteoarthrosis	207,000	216,000
Rheumatoid arthritis	71,000	92,000

This information which is based on private prescriptions for nonsteroidal anti-inflammatory drugs (NSAI) suggests that rheumatic disease is a common problem in medical practice. The data must be seen against the background of a population of nearly 24 million and the fact that 60-70% of the prescriptions are new.

There are very few groups who provide health care which have attempted to keep records of illness profiles. An examination of the records of the Anglo American Company's Medical Report provides further information about the nature and prevalence of rheumatic disease in Blacks. An examination of their latest report shows that morbidity rates for the various musculoskeletal diseases on the gold mines are; acute (non pyogenic) arthritis 0.39/1000, osteoarthritis 0.32/1000 and rheumatism (unspecified) 11.23/1000 employees. The data varies in the different mining groups, but the variations quoted may be more a reflection of classification rather than differing incidences. This is useful information but it provides a narrow view because it is concerned with the problem in Blacks only (Anglo-American Medical Department Annual Report 1980). An extraordinary lack of information exists in the Department of Health, Welfare and Pensions. In spite of its involvement in the provision of health care and welfare the department is unable to give any information about the disabilities for which it disburses funds. Very few medical aid societies seem to collect this type of information.

The population of South Africa is currently estimated at 23.8 million

(1980 census). It comprises people of several major groups - Negroid (16 million) and Caucasoid (4.45 million), Asiatic or Oriental (0.79 million) and a mixed or Coloured group (2.6 million) which has resulted from the miscegenation between the Negroid-Caucasoid and other groups (Botha and Pritchard 1972). From the genetic point of view the broad groups are as follows; Negroid (Negroid-Khoisan), Caucasoid, Coloured (Caucasoid-Khoisan-Negroid), Indian, Khoi-San (Jenkins 1972). This great diversity of peoples and cultures permits unique opportunities for studies in comparative medicine which may provide valuable information about the pathogenesis and natural history of disease. Few attempts have been made to look at the rheumatic diseases in this comparative way until recently. In the following pages the clinical and epidemiological studies which have been undertaken in South Africa will be reviewed.

A. INFLAMMATORY JOINT DISEASE

1. Clinical studies

The Southern African literature on the inflammatory arthropathies is sparse. Attempts to evaluate the prevalence and type of 'medical' arthritis were first reported by Gelfand who found 115 adult Black Zimbabweans over a 5-year period. The diseases which he encountered were gonococcal arthritis 16, septic arthritis 2, acute rheumatic fever 11, rheumatoid arthritis 11, acute non-specific polyarthritis 67, gout 6, ankylosing spondylitis 1, brucellosis 2, haemophilia 1 (Gelfand 1969). Very similar findings have been reported from Malawi in 90 patients seen during a three year period. Particular attention should

be paid to the few patients with rheumatoid arthritis (2) and gout (2) and to the high proportion of venereal related joint disease such as syphilis (9), gonococcal arthritis (3) and degenerative and traumatic joint disease (24). This report also drew attention to a nonspecific arthritis in 30 patients (Goodall 1956). The commonly encountered inflammatory rheumatic diseases of Black South African hospital patients are; rheumatic fever, tuberculous arthritis, rheumatoid arthritis, gonococcal arthritis, while Lupus erythematosus, gout and Reiter's syndrome are less common (Blumsohn 1976). The type of disease encountered by an orthopaedic service serving over 2 million Black South Africans are; bone/joint tuberculosis 35 cases/year, bone and joint sepsis 11 cases/year, rheumatoid arthritis 3 cases/year, juvenile chronic arthritis 2 cases/year, ankylosing spondylitis 2 cases/year and scleroderma 1 case/year (du Toit 1973).

1.1. Rheumatoid arthritis

The first clinical description of rheumatoid arthritis in Black South Africans was published in 1971 (Anderson 1971). In that study the milder course, remittent character and seropositivity of the disease was emphasised. The curious predilection for early ankle involvement, the general lack of serious deformity, or systemic manifestations were other features which characterised these patients.

Further attempts to define the characteristics of the disease were undertaken in the Transkei. In this study, three Mission hospitals contributed information for collation. There were 65 patients, of

whom 19 had definite rheumatoid arthritis (3 patients were under 16 years and were probably examples of juvenile chronic polyarthritis). The severity of the rheumatoid arthritis was graded as mild in 8, moderate in 4 and severe in 7, and the functional capacity of these patients, which was similarly graded, showed that there was mild impairment in 2, moderate in 10 and severe in 7. These subjects were mostly between 50 - 60 years (Percy-Lancaster 1974). The study gave no idea of the universe from which these subjects were drawn so that no conclusion about the prevalence can be made. The severity of the disease in Black South Africans has also been confirmed in another report (Chalmers and McNeill 1976). There are no comparable clinical studies of other peoples living in Southern Africa. There is little information about the social and personal impact of rheumatoid arthritis except for one study in White South Africans (Brighton and Louw 1981).

1.1.1. Juvenile chronic arthritis

A black female with Adult Stills disease (Naidoo 1981) and a child with Takayashu's arteritis and Stills disease (Hayes, Gwata, and Gelfand 1978) have been described. The literature on Juvenile Chronic arthritis (JCA) is equally sparse. There are very few reports dealing with this subject from the whole of Africa. The largest number have been reported from West Africa (Anumonye 1964) and although reference is made to six children with joint swelling in an early report from Malawi, the disease is not well described (Goodall 1956). Virtually nothing is known of the frequency of occurrence or the types of disease encountered. The disease occurs

in all of the three major race groups, and it is of some interest that uveitis in South African children with JCA is not associated with positive tests for anti-nuclear factor (Thomson, Bhettay and Steven 1976), but it seems that the disease does not differ in other ways from the described stereotypes (Bhettay and Thomson 1972).

1.1.2. Rheumatoid Factor

In a study of rheumatoid factor in 301 Black hospitalised non-arthritic medical patients in Durban, 21% had significant Latex agglutination titres which were related to increased levels of total seru, globulin and immunoglobulins. Liver disease was most commonly associated with significant elevated Latex tests (Chalmers, Pudifin and Shephard 1977). Using the sheep cell agglutination test (SCAT) this group has also reported that 90% of non-rheumatoid ill Blacks had a positive SCAT test (Chalmers, Pudifin, Shephard 1978). The prevalence of rheumatoid factor in the blood donor population of Cape Town was 1.13%, 1.05% and 2.15% for Caucasian, Coloured and Black Capetonians respectively (Meyers unpublished data).

1.1.3. HLA Tissue Typing in the Rheumatic Diseases

Tissue typing in rheumatoid arthritis has been investigated in Cape Town. In the Caucasoid rheumatoid arthritics strong positive associations have been shown for HLA DR4 49% vs 25% ($p= 0.001$) and DR3 37% vs 21% ($p= 0.004$). In the Cape Coloured DR4 49% vs 10% ($p= 0.0001$) was again significantly associated with

rheumatoid arthritis while in a smaller group of Blacks with rheumatoid arthritis it is even higher 60% vs 10% (p= 0.009). The data suggests that if a disease susceptibility gene for RA exists it is more closely linked with DR4 in South African Coloured and Black populations (Briggs, du Toit, Meyers 1980 unpublished data). HLA-DR4 gene frequencies are markedly different, approaching 0.2% for White and Coloured South Africans and less than 0.1% for Blacks, and the gene frequency for DR2 is very similar to this (Du Toit 1981). What significance these tissue types have in determining the prevalence of rheumatoid arthritis still needs to be clarified. It seems likely that the interaction of several genetic factors may be involved.

1.2. Ankylosing Spondylitis

Clinical descriptions of Ankylosing Spondylitis have been infrequent and there are sporadic reports of Black individuals with the disease (Gelfand 1969; Blumsohn 1976; Forbes 1960; Klemp and Meyers 1976; Chalmers, Seedat and Mudliar 1977; Chalmers 1980). The disease has also been infrequently reported in other parts of Africa. No clinical information is available in other population groups living in Southern Africa.

1.3. Systemic Lupus Erythematosus

There are two reports which deal with the clinical features of the disease, both of which have demonstrated no disparate features, when compared with world-wide series (Jessop and Meyers 1973; Seedat and Pudifin 1977). There are three reports from South Africa on

the familial occurrence of the disease (Agranat, Bersohn and Lewis 1957; Hift and Watson 1968; First 1973), and three Indian children with systemic lupus have been reported (Rovers and Coovadia 1981). In none of these reports is there a reference to Black males and it does seem as if the disease is particularly uncommon in this group. There is only one case report of severe aortic valve disease due to systemic lupus erythematosus (Thandroyen, Mattison and Weir 1978)- The skin problem of systemic lupus erythematosus is not a large one to judge from the small contribution which it makes to a Black dermatological practice (Schulz, Findlay and Scott 1962). One of the earliest descriptions of a lupus syndrome exacerbated by oral contraceptives was described from South Africa (Pimstone 1966). Mixed connective tissue disease was described in a short communication (Anderson and Anderson 1981).

1.4. Gout

Very few reports are available, from South Africa, and there is almost no information about the disease in the non Black populations. There are isolated clinical reports of the disease in Blacks and one is of particular historical importance because of its reference to the gout of Lobengula (Shepherd-Wilson and Gelfand 1962). It is a rare disease in Blacks, particularly rural Blacks, but others have however reported that gout is a frequent cause of polyarthritis in Blacks living in Zambia (Lowenthal and Diamond 1977). A classical Lesch-Nyhan Syndrome has been described in two white children (Schnier, Sims and Zail 1972) and a formes fruste of the Lesch-Nyhan in a Coloured

male (Cassidy, Gregory, Harley 1980). A female patient with hereditary xanthinuria and musculo-skeletal pain has been described with xanthine/hypoxanthinecrystals in her muscles (Berman 1975; Isaacs, Helfron, Berman et al 1972). This observation has important implications for the diffuse musculo-skeletal aching found in patients on Allopurinol therapy.

1.5. Psoriatic arthropathy

The arthritis associated with psoriasis has been studied in South Africa in a group of White and Coloured patients. The clinical pattern was no different in the two groups but the genetic pattern showed some differences. The previously claimed association between B23 + B17 were confirmed in those of Caucasoid origin while a different pattern A1/B8 was found in the Coloured South Africans (Green, Meyers, Gordon et al 1981). There are no accounts of psoriatic arthropathy in Blacks. An estimate of the frequency of psoriasis in Blacks suggest that it forms I.0 - 1.45% of a Black dermatological practice (Schulz, Findlay and Scott 1962).

1.6. Post rheumatic fever arthropathy

This rare arthropathy which follows acute rheumatic fever has been described in 7 patients, Caucasian, Coloured and Black (Meyers and Chalmers 1977) but it is surprising that so little has been seen in contrast to the high prevalence of rheumatic fever and chronic rheumatic heart disease in South Africans living in poor socio-economic circumstances (Chesler, Levin, du Plessis et al 1966).

Rheumatic fever in Blacks presents almost exclusively with carditis and arthritis (Schulze 1978).

1.7. Arthritis associated with infectious/infective illnesses

An outbreak of Chikungunya fever in a group of Caucasian adults and children has been described. The interest of this report lies in the chronicity of the joint disease, especially in adults, and in the strong association (4/5) of chronic joint disease with the tissue type B27. This study suggests that the chronic joint disease following Chikungunya fever is a form of reactive arthritis (Fourie and Morrison 1979). This is not a particularly widespread virus in South Africa (McIntosh, Seratini, Dickinson et al 1962). There is only one report of 3 patients who developed an arthritis associated with a preceeding rubella infection (Ritchken 1956). The arthritis associated with bacterial endocarditis has been studied in Caucasians, Coloured and Black South Africans. No differences were found in the three groups (Meyers and Commerford 1977).

Arthritis associated with infections with Yersinia enterocolitica have been described in South Africa and the 'rheumatic-fever like' clinical presentation has been emphasised (Forman and Kalk 1981, Thomas, Solomon and Rabson 1975). Attention has also been drawn to acute synovitis of the hip as a presenting feature of B.mellitensis infection (Eales 1951). Gonococcal arthritis has been infrequently recorded in the South African literature (Joyce-Clarke 1976; Hurwitz, Catchpole and Plit 1982). Twenty cases were collected over 2 years

in one Teaching hospital and the disease was categorised into acute, subacute and chronic forms. No data was available on the common dermal component of this syndrome. The diagnosis in the chronic form was based on the gonococcal complement fixation test which is an unreliable indication of gonococcal infections. It is possible that some of these patients may have had chlamydial infections. The paucity of reports is almost certainly due to under-reporting because venereal infections are frequent in Southern Africa. In a study in Black females from Zimbabwe the prevalence of gonorrhoea was 9.7% (Weissenberger, Robertson, Holland (1977). Little information exists about the arthritis associated with meningococcal infections. In one study of 57 Black miners with meningococcal infection, seen during one year, there were 2 (3.5%) patients with a pyogenic arthritis. Arthritis of the knee, elbow and the proximal interphalangeal joints is reported in South African Tick-bite fever. Attention was drawn in this report to severe back-ache which persisted for days after complete recovery (Elliott 1942).

An acute polyarthritis in Blacks has been recorded in Southern Africa which is characterised by fever, polyarthritis, leucocytosis and a benign course. No causative organism has been identified and no long term joint damage occurs. The illness is given different names such as acute non-specific arthritis, Congella arthritis (Gelfand 1963; Riley 1976). This is probably a viral infection although the evidence is scanty.

EPIDEMIOLOGICAL STUDIES OF INFLAMMATORY JOINT DISEASE

1. Rheumatoid Arthritis

There are several studies which have been undertaken to determine the prevalence of rheumatoid arthritis. The studies have been undertaken in rural and urban Blacks.

Rural Blacks

(a) Tswana Survey

The first study was conducted at the village of Phokeng 100 miles north-west of Johannesburg, (Beighton, Solomon, Valkenberg 1975).

(b) Xhosa survey

The second study in rural Blacks was conducted at St. Cuthbert's Mission at Tsolo in the Transkei (Meyers, Daynes and Beighton 1977).

Urban Blacks

The urban study was conducted in Soweto. This population was chosen deliberately, because it was culturally and genetically similar to the Phokeng villagers. The great majority of the inhabitants of this Township were either born there or had lived there for many years (Solomon, Robin and Valkenburg 1975). Similar investigative techniques were used in these three studies. The prevalence of rheumatoid arthritis was determined by the following modifications of the Rome Criteria (Kellgren, Jeffrey and Ball 1963).

1. Symmetrical arthritis/deformity of peripheral joints and especially of the metacarpophalangeal or metatarso-phalangeal joints, with involvement of at least one hand or foot.

2. Radiographic changes of grade 2 or more erosive arthritis. (Kellgren, Jeffrey and Ball 1963a).

3. A positive serological test for rheumatoid factor.

The modification which was made, was the omission of a history of morning stiffness, because of the difficulty which Blacks have with interpretation of this feature. Using these modified criteria a diagnosis of definite rheumatoid arthritis was made if three criteria were present, and of probable arthritis if two criteria were satisfied. In the rural studies, a diagnosis of clinical polyarthritis was recorded in 7 and 10 subjects respectively. Only one subject, in the second study, had classical rheumatoid arthritis, and in the other 16 the polyarthritis was mild and not easily recognisable as rheumatoid arthritis. In contrast, there were 24 respondents in the urban study with an inflammatory polyarthritis, which was more severe and included 2 subjects with advanced RA. Four subjects in the rural populations had definite rheumatoid arthritis and 5 subjects in the urban population had definite rheumatoid arthritis, which gives a prevalence of definite rheumatoid arthritis of 0.12%, 0.68% and 0.90% for the Tswana, Xhosa, and urban groups respectively. These studies have emphasised that

rheumatoid arthritis is much more readily recognisable in urban Blacks, and that the prevalence is strikingly different in rural vs urban living Blacks (p= 0.01). Rheumatoid factor was determined by the Latex agglutination (Latex) and human erythrocyte agglutination test (HEAT). In the rural subjects the prevalence of positive rheumatoid factor was 8.9% and 17.0% and 12.1% in the urban subjects. When the HEAT test was used the prevalence was 2.7%, 3.2% and 2.05% respectively.

2.2. Systemic Lupus Erythematosus

Systemic lupus erythematosus is much less easy to study in the field because it lacks the stereotyped patterns of rheumatoid arthritis. When the criteria for classification of SLE were proposed by the ARA, the hope was expressed that they would be useful in epidemiological studies (Cohen, Reynolds, Franklin et al 1971). There are however difficulties with the present criteria. Alopecia and mouth ulcers are not clearly defined, photosensitivity is difficult to evaluate historically and there are cogent reasons for the inclusion of anti-nuclear antibody (ANF) and serum complement profiles in the criteria (Weinstein, Bordwell and Rothfield 1978). In the absence of criteria which can be applied easily in the field, point case studies are the only indicators of the prevalence of the disease. In a retrospective study in Cape Town, the number of new cases presenting for admission to the medical wards was related to the number of other medical admissions. Thus, an admission rate was obtained for a population group for Caucasian, Coloured and Black females, this was 2.3, 4.9, 3.8/1000 respectively. A similar approach was used in Durban, where

a difference was also demonstrated for Indians and Blacks (Jessop and Meyers 1973; Seedat and Pudifin 1977). When the numbers of new cases over 10 years was related to the population of Cape Town (taken at the mid-point of the study), the prevalence was estimated as 5-6/100 000. It should be emphasised that this was an estimate based on the experience of one Teaching Hospital and it relied on sufficiently severe disease to warrant admission. It is therefore an under-estimate of the prevalence of systemic lupus.

2.3. Ankylosing Spondylitis

Studies in Caucasians have shown that the genetic endowment of B27 increases the risk of ankylosing spondylitis twentyfold (Calin and Fries 1975; Cohen, Nuttal, Schmid et al 1976). It is also of interest to draw attention to the small numbers of Blacks with ankylosing spondylitis who are B27 positive (Chalmers and Seedat 1977; Chalmers 1980) which may be a reflection of the low prevalence of HLA B27 in this group (du Toit and Botha; Hammond, Appadoo and Brain 1972). Based on the existing HLA B27 prevalence data in South Africans it is possible to predict the 'approximate number of people affected in South Africa. (Table 1.1.).

TABLE 1.2.

THE ESTIMATED PREVALENCE OF HLA B27 IN DIFFERENT RACE GROUPS IN SOUTH AFRICA

	NO: TESTED	PREVALENCE	1980 CENSUS (million)	ADULTS AT RISK (million)
Xhosa	240	0.83%)	15,97	0.005
Zulu	150	1.00%)		
Coloured	499	8.80%	2.55	0.009
Caucasoid	705	11.60%	4.45	0.051
Indian	147	1.00%	0.79	0.001

These estimates are only approximate and they assume that approximately 40% of the Black and Coloured population and 50% of the Caucasian and Indians are adults, and it also assumes that the published risk of 20% is applicable to all groups in South Africa. Only one subject with ankylosing spondylitis was encountered in the Tswana Survey, (Solomon, Beighton, Valkenburg et al 1975).

2.4. Hyperuricaemia and gout

No examples of clinical gout have been found in the epidemiology studies. Serum uric acid is low in rural (tribal) populations and is higher in the rural populations (Beighton, Solomon and Soskolne 1973; Beighton, Daynes and Soskolne 1976; Beighton, Solomon and Soskolne et al 1974; Beighton, Soskolne, Solomon et al 1974).

2.5. Pyrophosphate/Hydroxyapatite Deposition disease (Dieppe, Huskisson, Crocker et al 1976). There is little published information concerning chondrocalcinosis and its subsets, or of hydroxyapite related musculo-skeletal disease in South Africans (Schorn, Welke, Anderson 1975).

B. DEGENERATIVE JOINT DISEASE

There is one published clinical study on osteoarthrosis in South African Blacks. The clinical study documented the features of the disease and it drew attention to the low prevalence of hip involvement and the frequent knee involvement. Trauma was an important causative factor (Keen 1944). There has also been an epidemiological study in South African Blacks (Solomon, Beighton and Lawrence 1975) which has shown that the pattern of osteoarthrosis in the South African Black differs in several respects from comparable data in Caucasians. The disparaties between Black males and females for interphalangeal osteoarthritis are not as marked in Caucasians. Heberden's nodes were also relatively infrequent in Black females while knee osteoarthrosis was emphasised in the Black females. The reasons for the differences which were demonstrated, were discussed and they include occupational factors, obesity and mechanical factors such as walking barefoot. This study also drew attention to the lower prevalence of hip osteoarthritis in Blacks compared to Whites and it was suggested that anatomical abnormalities such as congenital dislocation of the hip, slipped capital epiphysis, minor abnormalities such as persistent femoral neck anteversion and acetabular dysplasia are rarely seen in South African Blacks. It could be that this is the reason for the disparity.

There are no comparable studies in other population groups in South Africa. A comparative study of intervertebral disc disease needing neurosurgical attention in adults from Zimbabwe showed that this type of disc disease was seen infrequently in Blacks when compared with Whites of a similar age (0.005/1000 vs 3/1000). Greater spinal mobility and spinal muscular development in Blacks was suggested to explain this difference (Levy 1969).

C. OSTEONECROSIS

There has been one study of this condition in Black South Africans over the age of 55 years in three communities - one rural, one urban and one semi-urban and a prevalence of 1.7% was measured (Solomon and Beighton 1975). The condition is reputed to be common in Coloured South Africans but no published data exists. The relationship to alcohol consumption is well-established (Jones, Jameson and Engelman 1968). In the Black subjects reported, a more complex relationship to alcohol and iron overload was suggested. One South African subject with avascular necrosis and hypothyroidism has been described (Seedat and Randeree 1975). The authors speculated on the possibility that the lipid disturbance of hypothyroidism was causally related. Lipid disturbances are associated with avascular necrosis (Murray-Leslie, Magaro and Wright 1976). A useful review of drug induced avascular necrosis of the femoral head in different race groups in Johannesburg has been published (Solomon 1973).

D. OSTEOPOROSIS

The only epidemiological work which has been reported has been almost

entirely on Black South Africans. An association between Bantu siderosis and spinal osteoporosis has been reported (Grusin and Kincaid-Smith 1954; Grusin and Samuel 1957; Seftel, Malkin, Schmaman et al 1966; Lategan 1971). Osteoporosis has also been studied in elderly Black and White South Africans in relation to fracture of the femoral neck. The reason for this study is the now well-known observation that Black females have a much lower femoral neck fracture rate than similarly aged White females (Solomon 1968). The studies have shown an essentially similar bone mass in both groups, so that the explanation for the discrepancy in fracture rate is not just due to different degrees of osteoporosis (Solomon 1979; Walker, Walker and Richardson 1971; Walker, Walker, Richardson et al 1970). There is a report of three Blacks with generalised osteoporosis and prominent periosteal reaction in long bones. The cause was not certain but a relationship to severe genito-urinary bilharzia was considered (Gelfand 1962). There was no evidence of osteomalacia or of hyperparathyroidism.

E. SARCOIDOSIS

Osteo-articular sacroidosis has been compared in Black, White and Coloured South Africans in the Western Cape. This study showed that the syndrome of erythema nodosum and arthralgia - arthritis occurred with nearly equal frequency in all three groups, but severe deforming disease was found only in the Blacks (Benatar 1980), and myopathy, as a presenting feature of the illness, was also more common in Blacks (Benatar 1977). Other reports on sarcoidosis in

South Africans also support the observation of a greater frequency of occurrence of osteoarticular disease in Blacks (Bowes and Jabkovitz 1952; Gordon Smith 1964; Weiss 1964) and in some it may be a particularly severe mutilating illness (Morrison 1974) resembling leprosy.

F. SCLERODERMA AND SCLERODERMATOUS DISEASES

There is a remarkable lack of information concerning scleroderma in South Africa particularly when one considers that the term progressive systemic sclerosis had its origin in South Africa (Goetz 1945), while the description of an increased incidence of scleroderma in gold miners was one of the earliest to relate scleroderma to occupational dust disease (Erasmus 1957). Scleroderma has been claimed to be the commonest Collagen-vascular in Black Zimbabweans (Gelfand 1970). A chronic form of scleroedema of Bushke which produced extensive joint contractures had been described in a Black female (Meyers and Quantock 1973), and there are now 3 patients reported with an eosinophilic fasciitis in the South African literature (Chalmers, Bhoola and Parboo 1979; Forman, Lewin, Gear et al 1981). The type and clinical course of polymyositis/dematomyositis has not been reported in the South African literature except in isolated case reports (Horsfall 1965; Sament, Klugman 1957; Findlay, Price and van Rensburg 1951). There is a report of a polymyositis syndrome in three members of a family, which proved to be due to typhoid. These three individuals were interesting because the illness presented like a myopathy (Naidoo and Chan Yan 1975)..

G. MSELENI JOINT DISEASE

This curious disease which occurs in the North eastern part of Zululand in the area around Lake Sibaya affects females more than males (39% vs 11%). It is predominantly a disease of the hips leading to progressive disability and problems with parturition. The condition resembles an epiphyseal dysplasia but the genetics of this condition are still in dispute (Du Toit 1979; Lockitch, Fellingham and Elphinstone 1973; Fellingham, Elphinstone and Whitman 1973; Lockitch, Fellingham, Whitman et al 1973; Nurse and Jenkins 1974). It bears a superficial resemblance to Kaschin-Beck disease and to Handigodu disease. There is also involvement of other parts of the skeleton. The ankle is the next most commonly clinically involved joint and there are radiological abnormalities in the lower radius/ulnar, (20%), shoulders and abnormally short metacarpals (10%). Recent investigations have suggested that a dietary mechanism may be implicated (Fincham, van Rensburg and Marasas 1981).

H. MISCELLANEOUS

There is not much published data concerning the fibromyalgic syndrome but it is claimed that this is a common problem in Black South Africans (Schulze 1973). Several other genetically determined diseases which have joint involvement as one of its features, have been described in South Africa. These diseases include Gauchers disease (Beighton and Sacks 1974; Goldblatt 1981), sclerosteosis (Truswell 1958; Falconer and Ryrie 1937; Beighton, Durr and Hamersma 1976), familial dyschondroplasia or Upington Disease (Schweitzer, Jones and Timme 1971).

There are two cases recorded of multicentric reticulo-histiocytosis in Black South Africans (Buchel 1970, Jessop and Gordon 1975). Another unusual hip disease affecting Coloured and Black prepubutal girls was described from Cape Town. The condition is called adolescent chondrolysis and it runs a subacute course with progressive narrowing of the hip joint and quite severe disability (Jones 1971; Sparks and Dall 1982).

Pyomyositis is a rare infection of skeletal muscles which is described from the tropics, but it has been described in subtropical parts of Southern Africa in the muscles of the back, chest wall, thigh and buttocks (Harari Staff Round 1971).

A strange arthropathy resembling Jaccoud's arthropathy has been described in association with familial cold urticaria (Commerford and Meyers 1977) and pachydermoperiostitis is recorded in a Black male (Findlay and Oosthuizen 1951). It is of interest that there are no reports of giant cell arteritis/polymyalgia rheumatica in Blacks. This may be because there are fewer Blacks who live long enough to develop the disease, and it may also be unrecognised because the symptoms are better tolerated. The genetic diseases occurring in Southern Africa, some of which are peculiar to South Africa, and which may have a rheumatological component, have been reviewed (Beighton, Durr and Hamersma 1976). Pagets disease of bone is unusual in South African Blacks (Robertson, Thomas 1976).

There are still considerable gaps in our knowledge of the occurrence of the rheumatic diseases in Southern Africa, and the gaps are even larger as far as the natural history of some form of rheumatic disease such as rheumatoid arthritis is concerned, while almost nothing is known of the personal cost or of the impact of rheumatic disease on individuals or the community in which they live. A body of knowledge is growing which will in time provide a fuller documentation of rheumatic disease in Southern Africa.

Epidemiological studies are costly and there is much to be said in favour of case collection and description, as pointers to further studies. The caveat that not all research comes via laboratories is useful to remember (Pijper 1934). Southern Africa is rapidly being industrialised, and urbanisation is increasing. If the urbanisation is important in the pathogenesis of rheumatoid arthritis, then we can expect to see it increasingly in Blacks in South Africa. Future comparative studies of rheumatic disease will doubtless be influenced by the growing importance of the genetic endowment of the host and important contributions have been and will continue to be made in this area by tissue immunologists. The rheumatic disease profiles of the South African Coloured community have hardly been explored, and the following study is a start in this direction.

CHAPTER 2

A BRIEF GLIMPSE OF HISTORY

Die Here het ga skommel en die dice
het verkeerd geval vi'ons daai's
maar al.
Adam Small 1936.

GEOGRAPHIC AND HISTORICAL BACKGROUND

GEOGRAPHY

Rietpoort (longitude 18S, latitude 29E) is the name given to an area of 15,092 hectares, 27 kilometers West of the Bitterfontein Railhead. It is a Coloured reservation area which has been occupied for over 130 years. The area lies in Namaqualand but it falls under the magisterial jurisdiction of Van Rhynsdorp. It is an arid part of the country lying at the foothills of the Kamiesberg to the North East, the Atlantic ocean to the West, and to the South and North are the Haardeveldt and greater Namaqualand. The average maximum rainfall is 50 mm/year. The geographic features resulting from the arid conditions and the landscape which is dotted with 'confused granite masses' makes this appear an inhospitable country.

The Coloured inhabitants live simple Western type lives, eking out an existence from a poor soil which they supplement with meat from a profusion of goats. The Community lives in three areas - Reitpoort - Putsekloof, Molsvlei - Stofkraal, and Lepelsfontein. These communities are administered by a Board of Management, but there are no features of town life such as public amenities, sanitation or piped water. Water is drawn from storage tanks and transported on foot or by car to the homes. The centre of activity is the Roman Catholic Mission which provides the cohesion for the community. There is a Church Hall where meetings are

held and the Mission also runs a General dealers store, Post office and a small hospital. Primary schooling is provided and most of the young adults find work on surrounding farms or on the Copper Mines.

HISTORICAL BACKGROUND

There was unprecedented political activity at the turn of the 18th century. Britain's supremacy at sea became established and the emotive words of liberty, equality and fraternity which fuelled the French Revolution were echoed over much of Europe. The Dutch East India Company controlled the Cape but it was increasingly concerned about British supremacy at sea and the 'dangerous' doctrines of the French Revolution.

Colonisation of the Cape Province

Since its establishment in 1652 the settlement at the Cape had gradually expanded beyond the confines of the Castle, although this was not company policy. A system of free burghers, many of whom were farmers and an increasing number of whom were stock farmers, had developed. In 1703 the first private grazing licences were issued without a specific demarcated 'farm' being set aside for this purpose. This soon developed into a system of loan farms which entitled a farmer to occupy an area of land temporarily. Initially these loan farms were restricted to owners of fixed property, but this fell away and any stock farmer could acquire a loan farm. This system more than anything else encouraged the development of the 'trek boer'. There were other

reasons too. The Company insisted on restrictive trade practices, and they discouraged association with the indigenous Hottentot and Bushman tribes and attempted to suppress the use of the French language by the French Hugenots. These were some of the factors which helped to encourage the movements of farmers away from the jurisdiction of the Govenor, and it was in this way that the Cape Province as far as the Orange River in the North and the Fish River in the east was colonised in the course of a century.[1.]

The origin of the Coloured people

The movement outwards into the interior brought the trek boers into increasing contact with the Hottentots and the Bushmen, and the intermingling between the predominantly Dutch Colonists with the Hottentots and slaves from West and East Africa produced the so called 'Bastard Hottentots' and these people came eventually to be called Coloured.[2.3.] The great diversity of physical characteristics of these people attests to their varying genetic background (Botha and Pritchard 1972).

The establishment and subsequent history of Rietpoort

As further waves of migration moved north-east, the indigenous people, the earlier settlers/trek boers were drawn further into

1. Standard encyclopaedia of Southern Africa Vol.10:p.626 et seq.
2. Standard encyclopaedia of Southern Africa Vol. 3:p.324 et seq.
3. Martin West, Divided Community. A.A. Balkema, Cape Town 1971.

the interior (some of them crossed the Orange River to avoid domination by the Colonial governors - these became known as the Oorlams Hotentots). In 1806 the Colony finally became British, whereafter more concerted attempts were made to bring some control over the indefinite frontiers of the colony. In the North West the local inhabitants petitioned the Crown for land which they could use and defend against the Trek boers who recognised no property or boundaries. As a result the areas of Concordia and Steinkopf were established by Sir Benjamin D'Urban in 1832 and on 21st February 1852 Sir Harry Smith granted three farms, Rietpoort, Stofkraal and Lepelsfontein to the 'bastard Hottentots' John Fredrick Owies, Christoffel Otta and Hendrick Epnaar in the magisterial district of Clan William (later Van Rhynsdorp). It appears that the early years of the usefruct of these three farms were uneventful, but by the end of the century Civil Commissioner Bam of Van Rhyndorp reported to the Surveyor General that 'the lands, especially Stofkraal, is occupied by a miserable class of natives growing up without education or religion' and the local veldtkornet reported that there were 17 members of the original family still alive and only three with families. The problem of the farms was further outlined by the Civil Commissioner as follows 'there is no recognised head, that each member of the family acts independantly of the others and each endeavours to get as much as possible out of the farm - without exertion, by letting pasturegrounds and lands for

sowing to the farmers residing in the vicinity.[1.]

The Civil Commissioner suggested that the tickets of occupation be revoked but nothing was done about this. It is claimed by some that these areas were later redefined as a reserve for the Coloured people in return for services rendered during the Boer war.[2.] Following the Boer war a more systematic control was exercised over the areas. In 1947 a special interdepartmental commission issued a report on the Coloured Mission Stations and Reserves.[3.] This commission evaluated the whole question of Coloured Mission Stations and amongst these were the three farms Rietpoort, Stofkraal and Lepelsfontein. It found that each of the three communities, Reitpoort, Stofkraal and Lepelsfontein had its own Board of Control which consisted of 2 corporals and 3 additional members. These officers served for as long as they rendered satisfactory service. Right of occupation was given to the descendants of the three original farmers (Owies, Epnaar and Otta) but newcomers were admitted after a period of probation. Part fulfillment was acceptance of the Roman Catholic faith. The privileges of the inhabitants was a grant of lands, the right to put up a building and pasturage. The affairs were run on dictatorial lines. At the time of the report is is clear that the inhabitants were not all descendants of the original families - in Rietpoort one third were not descendants and in Stofkraal there were 11 stranger families. Since the establishment of the independant department of Coloured Affairs, the administration of

1. Archival records SG 1/1/1/104.
2. Spore in die Dorsland. Gemsbok Drukkery Upington 1975.
3. Coloured Mission stations, reserves and settlements U.G. 33 1947.

the reserve became more firmly established. Today there is one management board for the three areas and there are plans to provide more facilities and establish a town at Rietpoort.

The role of the church

A very small N.G. Kerk community existed at Putsekloof from early times but the Roman Catholic Church put down its roots at Rietpoort with the arrival of Father Cornelius Van't Westeinde in May 1913.[1] In the face of great deprivation and strong local N.G. Kerk opposition this singular man proceeded to build the Mission. It is characteristic of the missionary that in spite of every kind of opposition he could say 'I am here and I am staying here'. Successive missionaries have worked there since, and the fruits of their labours are a successful mission station, schools, boarding establishment for 100 scholars and a well-equipped hospital. Although the Church no longer has official executive power in the affairs of the Community it continues to be intimately concerned with the welfare of the inhabitants and it still acts in an advisory capacity.

1. Spore in die Dorsland. Gemsbok Drukkery Upington 1975 p.19.

The area was chosen for an epidemiological study for several reasons :

1. THE PEOPLE WERE LIVING A SIMPLE RURAL LIFE AND THEIR LIFE STYLE WAS COMPARABLE TO OTHER RURAL COMMUNITIES WHICH HAVE BEEN STUDIED IN SOUTH AFRICA.

2. THE COMMUNITY WAS RELATIVELY STABLE.

3. THE ROMAN CATHOLIC MISSION PROVIDED SUITABLE PREMISES TO CONDUCT THE FIELD WORK.

CHAPTER 3

METHODOLOGY

When you cannot measure it, when you cannot express it in numbers, you have scarcely in your thoughts advanced to a stage of science whatever the matter may be.

Lord Kelvin 1824-1907.

METHODOLOGY

In July 1978 a census was taken of the inhabitants of the area by a door to door visit, during which the names of all the inhabitants were recorded. The people were advised of the proposed study and the Mission Staff helped by encouraging the people to attend. It was intended to use the whole adult population as the universe for the study. 3 months later the survey team started the field work on the premises of the Mission. The field work team consisted of 4 medical doctors, 3 nursing sisters, 2 radiographers, 1 laboratory technician and 2 lay helpers.

The mechanics of the field work was as follows:

ENTRY + REGISTRATION

COMPLETION OF QUESTIONNAIRE

EXAMINATION OF THE MUSCULO-SKELETAL SYSTEM/
DERMATOLOGICAL STUDY

HEIGHT, WEIGHT, ANTHROPOMORPHIC MEASUREMENTS

BLOOD COLLECTION

RADIOLOGICAL EXAMINATION OF HANDS/FEET

At the completion of the survey visits were made to the homes of the aged and infirm, who wished to be examined. Where possible they were taken to the Mission for their blood to be taken and for their radiographs.

1. The Questionnaire (See Appendix 1)

A questionnaire was completed by all the adults and the school

going children with the assistance of a trained Nursing Sister. The questions were directed at the following:

(1) Demographic data.

(2) Questions relating to chest disease/smoking habits.

(3) Previous/present backache, its duration, onset, associated spinal stiffness and the effect of exercise.

(4) Questions relating to arthritis - the presence of morning stiffness, joint swelling, the number of affected joints, the number of swollen joints, present or in the past.

(5) Previous or present shoulder pain.

(6) Questions about photosensitivity, alopecia, Raynaud's phenomenon, convulsions, pleurisy.

2. Clinical examination

The clinical examination included a record of the subject's height, weight and anthropomorphic measurements. Special attention was paid to the presence of features suggestive of Lupus erythematosus i.e. facial erythema discoid LE, thinning/fracturing of frontal hair, oral ulceration, pleurisy. The skin and nails were examined for evidence of psoriasis. Blood pressure readings were repeated several times if they were elevated and where possible the subject was asked to return the next day for further recordings. The degree, site and numbers of joints affected by arthritis was documented.

Hypermobility was measured using the following scheme:

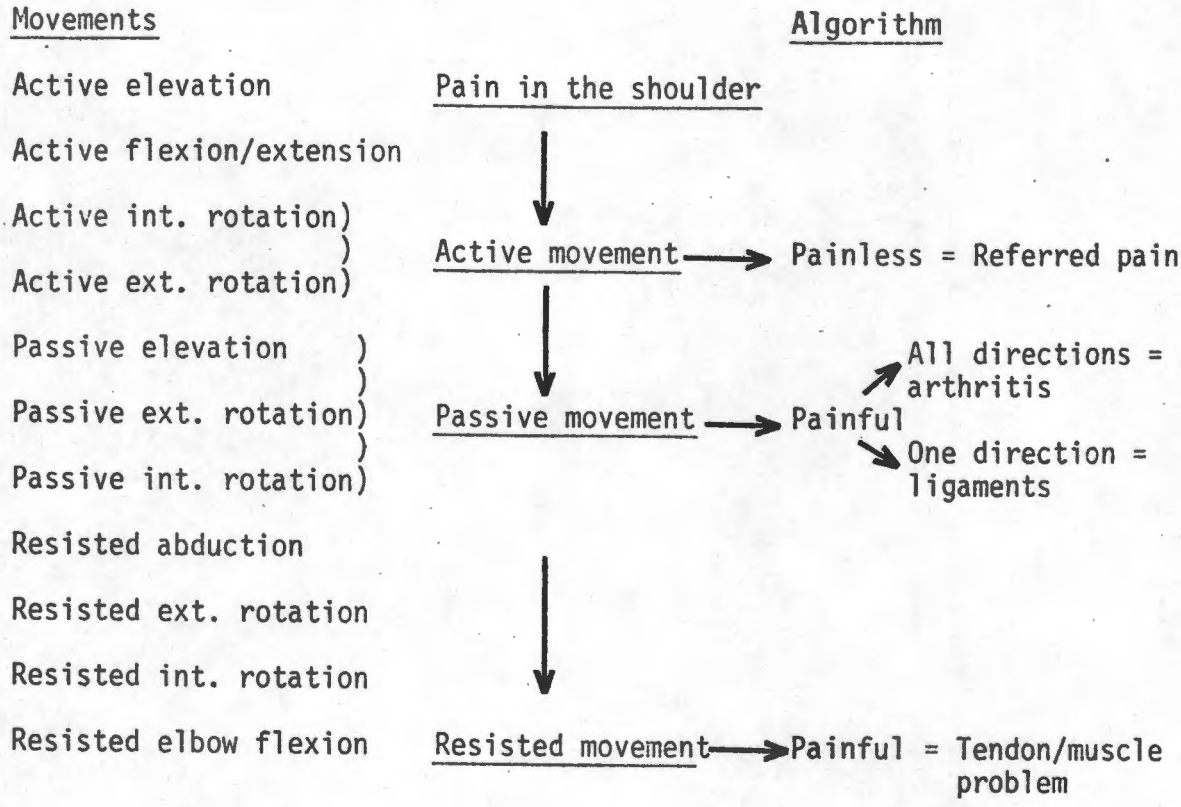
Passive dorsiflexion of little finger MCP joint to 90^{o} -	R+L	2
Passive apposition of thumb to forearm	R+L	2
Hyperextension of elbow beyond 10^{o}	R+L	2
Hyperextension of knee beyond 10^{o}	R+L	2
Forward flexion, palms flat on floor in front of the feet with knees extended		1
		9

Other soft tissue rheumatic complaints were also looked for.

2.1. Carpal tunnel syndrome was considered likely if there was symptomatic complaints of pain in the hand at night with numbness/ paraesthesia in median nerve distribution, with a positive Phalen sign.

2.2. Lateral epicondylitis was elicited by palpation of the lateral epicondyle for local tenderness, and associated pain at the epicondyle, and resisted dorsiflexion of the wrist with the elbow extended.

2.3. Shoulder problems were defined by the following algorithm (Chapter 8):



3. Laboratory tests

Blood was drawn for the following investigations:

3.1. Haemoglobin, white cell counts

Haemoglobin was measured using a standard colorimetric method in situ. White cell counts were done in situ with a standard haemocytometer.

3.2. Blood groups/serum iron/iron binding capacity/immunoglobulins

Blood groups were measured on heparinised blood which was transported in the cold to the laboratory in Cape Town. Serum iron and iron binding capacity were kindly performed in the Department of Chemical Pathology using a standard laboratory method. Serum immunoglobulins were measured in selected subjects with shoulder disease

using radial immuno diffusion. Commercially available plates were used (Hoechst).

3.3. Serum Complement profiles

Total haemolytic complement was measured in situ with a plate assay as described (Lachman, Hobart and Aston 1977). The technique is analogous to the Mancini technique for measuring immunoglobulins. Sheep cells sensitised to rabbit anti-sheep red cells were incorporated into 2% agar gels (Noble Agar). Equal volumes of sheep red blood cells and a commercial anti-sheep haemolysis (Difco Labs) were mixed and placed in a water bath at 42°C. Equal volumes of this mixture were added to 2% agar dissolved in complement buffer which was cooled to 42°C, and the mixture was well mixed. 2 mm thick plates were poured in Petri dishes. After gel setting occurred wells were punched in the agar. The plates were stored at 4°C until further use. 10 micro litre of test serum was dispensed into the wells and control sera whose complement levels were previously determined were included in each plate as controls. The control serum was stored in aliquots which were kept frozen until used. A fresh aliquot was used each day. The sera were allowed to diffuse into the agar overnight at 4°C. On the following day the plates were incubated at 37°C for 1 hour. The lysis around each well was read and the results were read from a standard curve prepared in the Laboratory. The method was tested in the Laboratory prior to the field study and compared with a standard tube assay for total haemolytic complement (Kent and Fife). In a comparative study of 20 sera, the two methods yielded

means of 189.25±12.5 units/ml and 195.25±12.6 units/ml for the plate and tube methods respectively. The difference between the two methods was not significant ($p \geq 0.500$). Serum C3/C4 was measured in the laboratory on stored serum using immunoplates kindly donated by Hoechst (Pty) Ltd. The sera which had total haemolytic complements of less than 100 units/ml were tested in the laboratory for their C3/C4 content, in order to determine whether the low total complement was due to early complement fraction consumption.

3.4. Rheumatoid factor

Rheumatoid factor was measured by two methods, the Human Latex agglutination test and the Human erythrocyte agglutination test.

3.4.1. Human Latex Test

All the sera were screened in the laboratory using a Standardised Latex RA kit (Ortho) which was kindly supplied by Ethnor Laboratories. All the positive sera were then tested by a standardised tube method (Singer 1974) to determine the titres of rheumatoid factor.

3.4.2. Human erythrocyte agglutination test

This test was carried out as described by Valkenburg on the sera in the laboratory. The anti-serum to human O cells raised in rabbits was used in a previous study (Meyers, Daynes and Beighton 1977).

3.5.0. Other Human auto-antibodies

Human auto-antibodies were looked for in stored sera using a standard sandwich immunofluorescent technique and a range of tissues as sub-

strates for anti-nuclear antibody, anti-smooth muscle antibody and human kidney for anti-mitochondrial antibody (Friou 1957; Whittingham and MacKay 1969; Handbook of Techniques W.H.O.). Human anti-thyroid auto antibodies were measured using a kit for anti-thyroglobulin and anti-microsomal antibody. This method is based on a formalised tanned sheep red cell agglutination which is widely used (Wellcome Labs. Product information).

3.5.1. Antibodies to extractable nuclear antigen (ENA)

A commercial preparation of calf thymus extractable nuclear antigen (ENA) (Pel Freeze Batch No 3433) was used as the antigen. Concentrations of 0.15 mg/ml dissolved in McIlvaines buffer pH 7.2 were used to coat tanned, formalinised human O cells. Human O cells from 20 donors were formalinised as described (Herbert 1973). The cells were well washed and stored for at least 2 weeks at $4^{o}C$ in McIlvaines buffer pH 7.2. Tanning of the cells was carried out using a concentration of 5 mg/100 ml freshly prepared Reagent grade Tannic acid (Merck) as described (Herbert 1973). The antisera were diluted 1:20 in microtitre plates and serial doubling dilutions to 1:160 were prepared using McIlvaines buffer as diluent. After the addition of the coated O cells, the plates were tapped gently to mix the cells. Controls of coated cells with diluent, and uncoated cells with antiserum were included. The prepared plates were left at room temperature and read 18 hours later. The agglutination pattern was read using the following grading: 4+ a smooth mat of cells, 3+ a smooth mat of cells surrounded by a thin irregular rim of packed cells, 2+ a smaller mat with a broader irregular rim of packed cells, 1+ a dense

irregular rim of cells, O a tight smooth button of cells. A 2+ reaction was taken as the end point. The positive sera were diluted further to determine the exact end point and all positive sera were tested against RNAse treated ENA coated, O cells. Two and a half millig rams RNAse/ml (Miles Laboratories) was dissolved in phosphate buffered saline and added to ENA coated cells. The mixture was incubated for 30 minutes at 37°C. Following the incubation the cells were washed three times in buffered saline before being added to the serial dilutions. The same grading was used to determine the end point. All the sera were also tested by counter immuno electrophoresis as described (Bresnihan, Bunn, Snaith et al 1977).

3.6. Serological tests for syphilis

Two tests were used, the VDRL and the TPHA. Both these routine tests were kindly done by the Department of Bacteriology at UCT.

3.7. Serum uric acid

Serum urate was measured in the Department of Chemical Pathology using an autoanalyser technique. Appropriate controls were included in each batch. The autoanalyser was used because of the large number of specimens. Autoanalyser measurements give higher readings than the uricase method; the levels are 0.2 mg./100 ml. and 0.14 mg/ml. higher in males and females respectively with the automated method (O'Sullivan, Francis and Kantor 1965). Part of the increased concentration of uric acid with the auto-analyser method is believed to be due to chromogens which dis-

appear after 3 months of storage (Buchanan, Isdale and Rose 1965). The Namaqualand sera were all measured on serum stored for 3 months.

3.8. Serum proteins/protein electrophoresis

Total serum protein was measured with the Biuret method and all sera were then electrophoresed using a standard cellulose acetate strip (Beckman microzone). Serum albumin levels were computed from the total protein-electrophoresis strip.

4.0. Urine examination

All the adults in the survey provided a specimen of urine which was tested chemically using Ames Multistix and the urinary sediment was examined microscopically.

5.0. Radiological examination

Radiographs of hands and feet were taken of the adults over 15 years. Radiographs were taken at a fixed tube to film distance of 39 inches, using Standard X-ray film. The films were processed in situ. All the radiographs were read at completion of this survey.

5.1. Radiographs were evaluated for evidence of erosive arthritis using the standard atlas of radiographs (Kellgren, Jeffrey and Ball (1963a).

5.2. Osteoporosis was measured using the area index. This was derived from measurements of the mid point of the second right

metacarpal. The area index was standardised by obtaining the ratio of area index/surface area (see chapter 9 for details).

5.3. Osteoarthrosis was determined by the following grading system:

Normal	= 0
Suggestive osteophyte	= 1
Definite osteophyte (normal joint space)	= 2
Decreased joint space with osteophytes	= 3
Disorganisation of the joint (joint space, narrowing, subchondral cysts, deformity)	= 4

The proximal and the distal interphalangeal joints, the metacarpal-phalangeal joints and the carpometacarpal joint of the thumbs were examined and graded for osteoarthrosis. In the feet the metatarsophalangeal joint of the big toe was graded in the same way (Lawrence 1977g).

5.4. Hallux Valgus was measured as shown on the diagram: (Figure 3.5.4.)

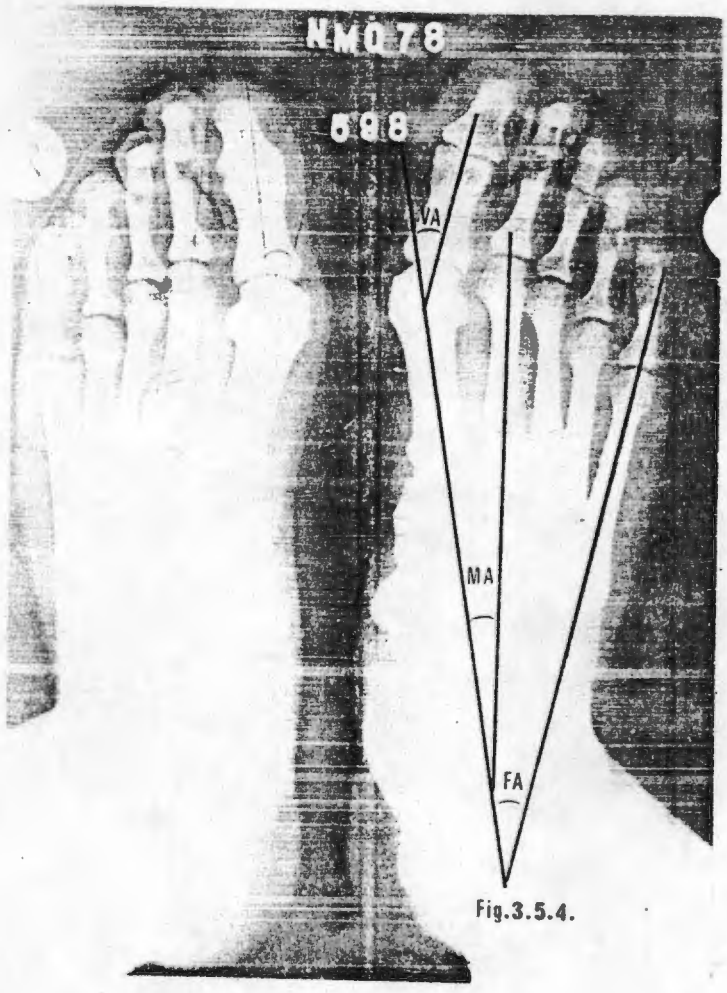


Fig.3.5.4.

The first, second and the 5th metatarsal of both feet were bisected along their length at their mid point and the lines were continued until they bisected each other. The angle between 1st and 2nd metatarsal described the intermetatarsal angle, and that between the 1st and 5th as the forefoot angle. The hallux angle was described where the line drawn through the length of the proximal phalanx of the big toe at its mid point bisected the line drawn through the 1st metatarsal.

The statistical evaluations where necessary used either the Student's t test or the X^2 test.

The results of some of the clinical evaluations of the haemoglobin, white cell counts and urine examinations will not be reported in this thesis because they form part of a study on systemic lupus erythematosus to be reported elsewhere.

The age specific prevalence is shown in decades and it follows a standard format 15 - 24 years, 25 - 34 years etc., up to 65 - 74 years. In a few instances the data is analysed into a further decade i.e. 75+. This was done particularly for measurements of bone density.

CHAPTER 4

DEMOGRAPHY

DEMOGRAPHY

4.1.0. Census

The census which was undertaken counted 1637 inhabitants. Their age and sex distribution is shown in Table 4.1.

TABLE 4.1.1.

AGE AND SEX DISTRIBUTION OF THE SURVEY POPULATION

AREA:	SEX	0-14	15-24	25-34	35-44	45-54	55-64	65+
RIETPOORT	Male	184	58	34	33	29	16	37
	Female	177	75	48	26	26	30	39
MOLSVLEI	Male	104	42	21	22	18	14	24
	Female	126	62	30	28	12	13	18
LEPELSFONTEIN	Male	74	30	8	13	13	10	15
	Female	55	23	11	13	12	4	10
	TOTAL:	720	290	152	135	110	87	143 = 1637

TOTAL MALES = 799

TOTAL FEMALES = 838

The census data agrees with the information which is contained in the annual report of the management board for 1977 in which the number of inhabitants is given as 1665. This close agreement between the census and the annual report provides a validation for the method and the

Thoroughness of the census. The census enumerator only documented people living in the areas permanently, and excluded those working away. This may account for the difference between the census and that of the management board.

4.2. Completion rates

The number of persons seen during the survey is shown in Table 4.2.1.

TABLE 4.2.1.

NUMBER OF SUBJECTS SEEN DURING THE FIELD WORK - BY SEX AND AGE

AREA:	SEX	0-14	15-24	25-34	35-44	45-54	55-64	65+
REITPOORT	Males	127	42	24	26	23	14	33
	Females	154	55	48	28	25	25	33
MOLSVLEI	Males	80	28	17	13	11	12	19
	Females	89	39	25	22	22	12	18
LEPELSFONTEIN	Males	51	22	8	12	10	7	14
	Females	39	12	5	12	13	4	10
	TOTAL:	541	198	127	113	95	74	127 = 1275

TOTAL MALES SEEN = 594 (Males over 15 years = 335)

TOTAL FEMALES SEEN = 681 (Females over 15 years = 399)

The completion rate for this population was 77.9% and for the adults over the age of 15 years it was 80.04%. In the 3 areas the completion rate for adults was; Rietpoort 84.49%; Molsvlei 75.32%; and Lepelsfontein 79.62%. The easier access of the Rietpoort inhabitants to the Mission is considered to be the reason for the highest completion rate. The completion rate for children was 75.13%. Blood was drawn from 652 of the 734 individuals over the age of 15 years. This represents a completion rate of 89%. A total of 599 hand and 595 foot radiographs were taken which represents a completion rate of 81.6%.

4.3. Residential features, occupation and income

There were 30 permanent brick houses in the area. Other homes were made of corrugated iron and mud bricks. The average number of persons living together was as follows:

71% more than 5 persons/house
10% 4 persons/house
8% 3 persons/house
6% 2 persons/house
5% 1 person/house

The houses varied considerably in size, the permanent houses being the largest. Three quarters of the houses were 1-2 roomed and one fifth were 3 roomed. The houses occurred alone or in small clusters and they were separated from other houses by some distance.

The occupation of the inhabitants is given in the following Table 4.3.1.

TABLE 4.3.1.

OCCUPATION OF INHABITANTS

OCCUPATION	%
Unemployed (includes housewives)	49.7
Labourers	24.9
Pensioners/Disabled	14.4
Builders/Painters	1.2
Teachers	2.5
Domestic workers	2.8
Farmers	0.7
Other	3.8

The average income varied considerably but it was generally low. 2.0% earned less than R20/month, 30% earned R20-50/month. 43% earned between R50-100/month and 25% earned more than R100/month. None of the households had piped water. The population depends upon rain water tanks, and mist/dew which precipitates on the granite outcrops, which is channeled into large storage tanks. Sanitation was provided in the form of either a bucket or pit system. The staple food was wheat supplemented with meat and some vegetables. The arid conditions do not allow for cultivation of vegetables on any scale. Many of the males drank cheap wine at weekends.

4.4. Height and Weight

The population were relatively short statured. The height and weights are shown in the following Tables Table 4.4.1. and Table 4.4.2.

TABLE 4.4.1.

HEIGHT OF MALES AND FEMALES IN NAMAQUALAND (METERS)

	15+	25+	35+	45+	55+	65+	75+
MALES	1.66±0.03	1.65±0.01	1.65±0.01	1.66±0.03	1.64±0.01	1.67±0.01	1.59±0.02
FEMALES	1.53±0.03	1.53±0.03	1.56±0.03	1.55±0.03	1.52±0.01	1.52±0.01	1.51±0.01

TABLE 4.4.2.

BODYMASS OF MALES AND FEMALES IN NAMAQUALAND (KG)

	15+	25+	35+	45+	55+	65+	75+
MALES	55.9±0.8	57.4±1.1	57.4±1.2	58.8±1.5	57.1±1.7	59.3±3.2	54.2±2.6
FEMALES	51.9±1.0	51.7±1.1	55.6±1.8	54.5±2.0	54.3±2.3	59.2±3.1	50.9±6.2

The mean height for males and females is 1.64±0.017 metres and 1.53±0.045 meters in females. The mean height for the females in this population varies by one centimetre from the mean height reported in 1,186 Coloured primigravida from Cape Town (Woods, de V. Heese, Davey et al 1978), but the post-delivery weights are much higher than the mean weight for the Namaqualand females. The mean mass for males and females was 57.14±0.66 and 54.02±1.08 kg. respectively. Two standard deviation was used to

define the limits of normality. The ranges were 55.85 - 58.43 kg. for males and 51.17 - 56.87 kg. for females. The lean body mass was calculated from the formula of Siri (Siri 1961) and is shown in the following Table 4.4.3.

TABLE 4.4.3.

LEAN BODY MASS IN MALES AND FEMALES (Kg.)

	15+	25+	35+	45+	55+	65+	75+
MALES	49.7±1.0	51.0±0.9	51.3±0.8	51.0±1.1	49.0±0.9	50.9±1.5	45.2±1.6
FEMALES	38.9±0.5	28.8±0.6	41.5±0.9	40.5±1.1	40.0±1.3	41.1±1.6	39.0±3.3

The mean lean body mass for males and females was 49.65±0.80 and 39.87±0.43. Two standard deviations were used to define the limits of normality for this population. The range was 45.45 - 53.85 and 37.67 - 42.07 kg. for males and females respectively. Using these defined limits 19.6% of the males and 38.8% of the females had a lean body mass below normal.

The nutritional status of the population was good and no examples of overt nutritional deficiency was found. The serum albumin levels were also measured to assess nutritional status. This information is presented in the following Table 4.4.4.

TABLE 4.4.4.

MEAN SERUM ALBUMIN LEVELS IN MALES AND FEMALES (Gm/L)

	15+	25+	35+	45+	55+	65+	75+
MALES	39±0.5	39±0.4	38±0.6	37±0.5	39±1.3	36±0.9	34±0.7
FEMALES	36±0.4	37±0.4	36±0.9	36±0.6	37±0.9	35±0.7	32±1.2

The serum albumin levels varied slightly in males and females up to the age of 65 years whereafter the mean levels dropped so that in the subjects over 75 years the levels in both the males and the females was below 35 gm/litre. Levels of serum albumin below 35 gm/litre are used as evidence of malnutrition provided there is no other explanation such as chronic liver disease, rheumatoid arthritis etc (Davidson, Passmore and Brock 1975). None of the elderly subjects had evidence to suggest chronic liver disease clinically and the subjects with rheumatoid arthritis were not included in the analysis. The measurement of lean body mass has been difficult to measure without special equipment and techniques. A great deal of ingenuity has been shown by many people in divising anthropometric formulae for measuring lean body mass. The results have proved disappointing not because the problem is insoluble, but because measurements of lean body mass is difficult. The measurement of fat content of the body has been somewhat easier because more positive relationships have been shown between skinfold thickness and total body fat (Brozek, Keys 1951; Consolazio, Johnson and Pecora 1963). The Namaqualand subjects were generally lean and there were very few subjects who were considered to be obese. The lean body mass was calculated to be 83-89% in males and 69-75% in females.

THE RIETPOORT MISSION

THE ENVIRONS OF THE MISSION

SOME OF THE CHILDREN OF RIETPOORT

AN ELDERLY MEMBER OF RIETPOORT

WATER IS CHANNELLED FROM THE GRANITE
OUTCROPS INTO STORAGE TANKS

COLLECTING HOUSEHOLD WATER

THE HOUSES OCCUR IN SMALL CLUSTERS

THE HOMES ARE SIMPLE WITH NONE OF THE
AMENITIES OF A TOWN/CITY

STARTING THE DAY

THE M.R.C. MOBILE X-RAY/LABORATORY

WAITING FOR THE X-RAY

THE ARTHRITIS FOUNDATION COMBI WAS USED TO
FETCH THE ELDERLY AND INFIRM

CHAPTER 5

PREVIOUS HISTORY OF ARTHRITIS AND/OR BACKACHE

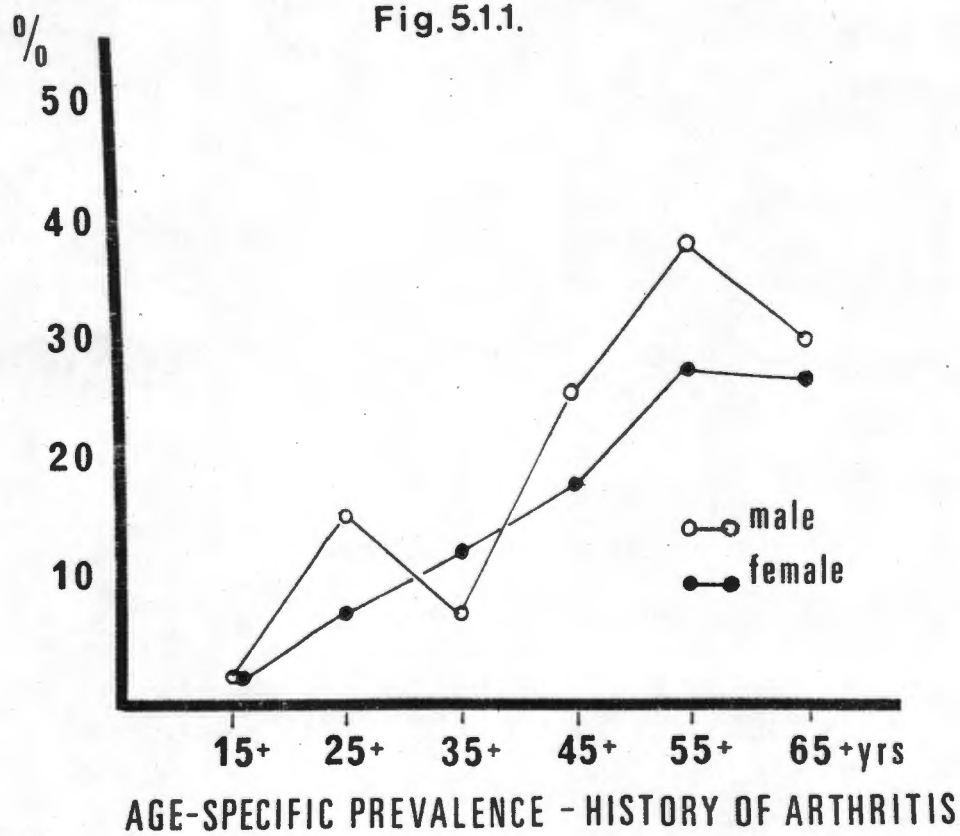
For still the more he works the more do
his weak ankles swell. Simon Lee.
Wordsworth. 1770 - 1850.

PREVIOUS HISTORY OF ARTHRITIS AND/OR BACKACHE

5.1. Previous history of arthritis

Sixty-one (61) males and forty-eight (48) females recalled a previous arthritis history on one or more occasions. This represents a prevalence of 18.2% for the 335 adult males, and 13.8% for the 399 adult females. The age specific prevalence for the males and females is shown in Figure 5.1.1.

Fig. 5.1.1.



AGE-SPECIFIC PREVALENCE - HISTORY OF ARTHRITIS

In the males there were two peaks; one at 25 - 34 years and a second at 55 - 64 years. In contrast the females showed a progressively

increasing prevalence which reached a peak at 55-64 years. The data was analysed further in 2 groups; those aged 15-44 years (15) and others aged 45-65+ years (46). In the younger males there were two with a polyarthritis history, 6 with a history of 2-4 joint involvement, and 5 with a single joint arthritis. In two the number of affected joints was not specified. The affected joints were hands 5, feet 3, elbows 2, knees, shoulders and ankles 1 each.

In the older males, 10 subjects reported a polyarthritis, twentythree had 2-4 joints involved, twelve had 1 joint involved. One subject did not specify the number of affected joints. In this group the knees were most commonly involved followed by the hands, ankles and elbows. There was an increasing number of subjects with radiological evidence of osteoarthritis in their hands/feet in the older males so that at least some of the arthritis recalled by this age group can have been caused by osteoarthrosis. Since the questionnaire did not attempt to define what each individual meant by arthritis, it would be reasonable to assume that in some of the individuals arthritis probably means joint pain without an inflammatory component.

In the younger females (13) the hands were involved in four, the feet in three and in six the site was unspecified. In this group, one (1) reported a polyarthritis, 8 involvement of 2-4 joints and 2 reported involvement of one joint, and in 2 it was not specified. In the older females (35) the knees and hands were again most commonly reported. In this group 4 were polyarthritic, 17 reported 2-4 joint

involvement, 11 reported a mono-arthritis. The data is summarised in the following Table 5.1.1.

TABLE 5.1.1.

DISTRIBUTION OF JOINT INVOLVEMENT (BY HISTORY)

	15 - 44 YRS		45 - 65+ YRS	
JOINT GROUPS	MALES(15)	FEMALES (13)	MALES(46)	FEMALES(35)
HAND	5	4	15	11
FOOT	3	3	5	6
KNEE	1	1	19	14
SHOULDER	1	-	2	1
ANKLE	1	1	5	2
ELBOW	2	-	6	2
HIP	-	-	3	0
UNSPECIFIED	3	6	14	14

The prevalence of polyarthritis was 3.58% in males and 1.25% in females and 2.3% for the whole adult population.

LACK OF CLINICAL RESIDUA

The clinical residua of these previous episodes of arthritis are not easily assessed because not all the joints which were reported to be involved were examined carefully to determine whether there was a reduction in range of motion, or premature degenerative joint disease. In the shoulder such an evaluation was done in all individuals (see

chapter 8). There were 24 subjects in whom there was a reduced range of motion in one or more directions at the shoulder. In 16 of these there was a previous history of shoulder pain some of which is presumed to have been due to shoulder arthritis. In eleven other individuals a decreased range of motion of a joint or group of joints was recorded. This was most commonly in the hands/feet/wrists. These subjects were all elderly (for further discussion see chapter 6). The radiographs were not taken of all previously affected joints but the radiographs of the hand and foot of those with a positive history of arthritis affecting hands or feet did not show evidence of prior joint damage. None of these subjects had positive rheumatoid factors.

THE CAUSE(S) OF THIS SYNDROME

In other surveys of rheumatic disease, 2-4% of individuals have reported an episode(s) of polyarthritis. The historical data suggests that this is an acute, transient arthritis which commonly starts in the feet, spreading to ankles, knees and hands. There is also sometimes mention of severe pain in the axial skeleton suggesting facet joint involvement. The true frequency of this recall of arthritis is probably much higher than the prevalence rates suggest because some of the transient joint pain may be forgotten. The term benign polyarthritis is applied to this historical syndrome (Lawrence 1977j). It has been regarded by some to be a mild form of rheumatoid arthritis, but this is not a good explanation because if this were so, it would be expected

That this symptom complex would occur more frequently in the histories of patients with definite rheumatoid arthritis, which is not the case. Furthermore none of the other attributes of rheumatoid arthritis have been found in the subjects with benign polyarthritis. Others have suggested that these are episodes of acute rheumatic fever, but there are again good reasons for not accepting this possibility; notably because there is a disparity between the occurrence of the two diagnoses in population studies. It needs to be emphasised however that the diagnosis of a poly-arthritic illness as rheumatic fever in adults is not easy because of the relative absence of carditis (Davis 1970; Ben-Dov and Berry 1980) and the known difficulty in establishing evidence of present or previous streptococcal infection (Kaplan, Top, Dudding et al 1971; Bisno and Stollerman 1975). Another possibility is that the syndrome represents the peripheral manifestations of inflammatory spondylitis (Hollister and Engelman 1958), or Juvenile Chronic Arthritis (Stills disease). In Britain there is a seasonal incidence for this disease, and it is highest in the winter months (Lawrence 1977a). Others have also claimed a relationship to upper respiratory infection, streptococcal and others (Abdin 1971; Greenwood 1969a). No attempt was made in the Namaqualand population to relate the polyarthritis to seasons of the year.

An examination of the literature suggests that this is probably not a single diagnosis and that many infectious agents can reproduce this syndrome. The agents which have been implicated are viruses such as the arbor viruses causing O'nyong O'nyong, Ross river virus,

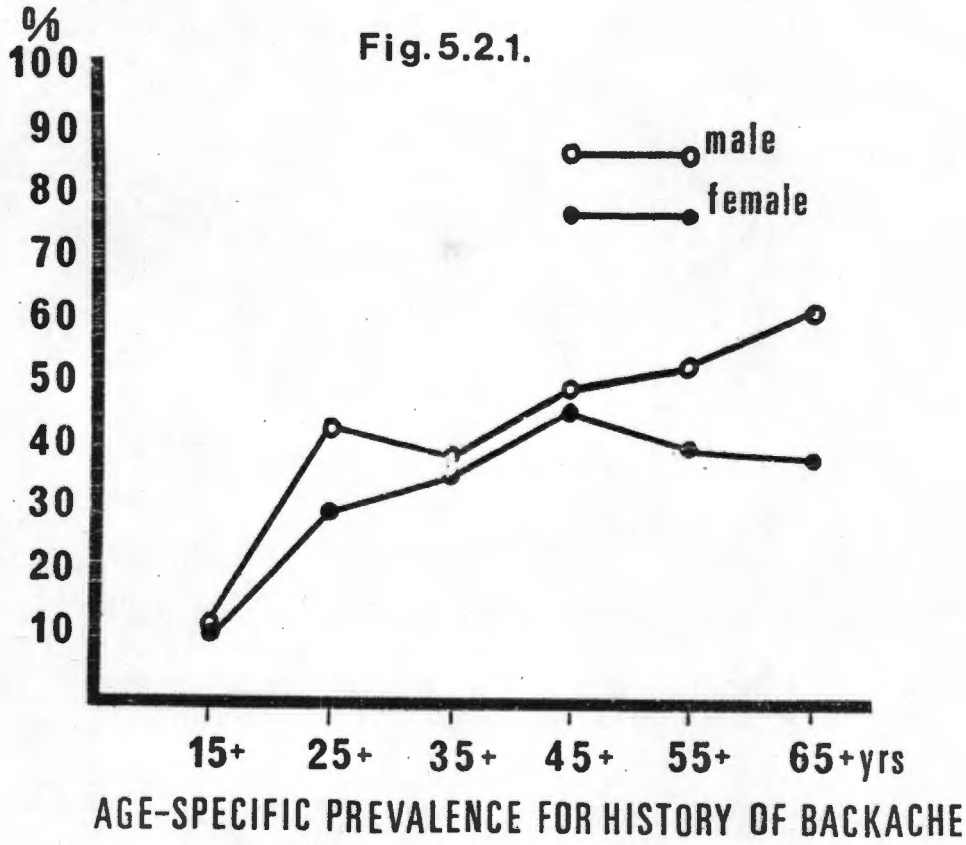
Chikungunya (Smith and Sandford 1967), Rubella virus and Hepatitis B virus (Hyer and Gottlieb 1978) as well as Mycoplasmas (Lambert 1968). Attention has also been drawn to infections with Yersinia enterocolitica, as a cause of polyarthritis which mimics acute rheumatic fever (Leivisalo and Laitinen 1975; Forman and Kalk 1981). In Southern Africa the acute non-specific polyarthritis of Blacks which resembles an acute tropical arthritis described in Nigeria (Greenwood 1969a) may also make a contribution to benign polyarthritis, but this is of course not relevant to the Namaqualand population. Although benign polyarthritis is not considered to be part of the syndrome of rheumatoid arthritis there are some who believe that in those subjects where the cause of the polyarthritis is undertermined it may represent a benign form of rheumatoid arthritis. Such an idea was suggested by Glynn (Glynn 1968) who proposed that the clinical disease rheumatoid arthritis is a benign polyarthritic illness on to which has been grafted an auto-immune reaction. There are considerable difficulties in proving such an hypothesis. The cause for the episodes of pauci-articular 'arthritis' i.e. 2-4 joints or mono-articular 'arthritis' in this Namaqualand population is likewise uncertain. Many of the putative infections or infective agents discussed above can produce pauci-articular disease. Serological tests for syphilis were present in 9.62% of this population and it may be assumed that venereal infectious agents could also have produced some of the episodes of mono or pauci-articular episodes (see chapter 6).

5.2. Previous history of backache

Backache was a prominent feature in the population of Namaqualand.

One hundred and thirty-nine males (139) and one hundred and twenty-eight females (128) had backache or recalled previous backache. This represents a prevalence of 36.5% (41.3% males, 32.0% females). The age specific prevalence is shown in the following Figure 5.2.1.

Fig.5.2.1.



AGE-SPECIFIC PREVALENCE FOR HISTORY OF BACKACHE

In males there was a peak recall of backache in the 25-34 year age group and thereafter a progressive rise in the historical recall of backache to reach levels of 60% in the subjects aged 65+ years. In females there was a steady rise in the prevalence of recalled backache to 49% in those subjects aged 45-54 years, whereafter the curve declined. It is of interest that the first peak of backache in males

aged 25-34 years occurs at the same time as historical recall for previous arthritis while the graph for the females retains the same curve as for the arthritis history in females. It is impossible in retrospect to derive much more useful clinical information from the data, but the coincidence of the of the two curves for arthritis/backache in males suggests that the two may be related. The clinical pattern of benign polyarthritis has already been detailed, and it seems likely that these curves are describing the occurrence of this condition in young males. This is however only one possible explanation for the backache in the young individuals and a variety of conditions such as osteochondritis, ligamentous injury and disc prolapse can also contribute to this peak of backache. In the older individuals degenerative spondylosis together with osteoporosis would also contribute to the increasing frequency of backache (see chapter 7 and chapter 9). The symptoms profile of backache in this community is shown in the following Table 5.2.1.

TABLE 5.2.1.

SYMPTOM PROFILE OF BACKACHE

	MALES	FEMALES
Onset - Insidious	66%	87%
- Acute	34%	13%
Duration (3 months)	74%	71%
Associated spinal stiffness in the morning	84%	75%
Improvement with exercise	71%	74%

The historical features which suggest the presence of inflammatory backache are:

1. An insidious onset
2. Age under 40 years
3. Duration for longer than 3 months
4. Associated spinal stiffness in the morning
5. Improvement of symptoms with exercise

This combination of symptoms has been shown to be 95% sensitive and 85% specific for inflammatory backache (Calin, Porta, Fries et al 1977). In the Namaqualand population 21 subjects under the age of 40 years gave this characteristic history (for further discussion see chapter 6). The greater number of males with an acute onset of backache is probably the result of their occupation. No attempts were made to measure the degree of lumbar lordosis in these people, but it is important to consider that part of their origin is Khoisan i.e. Bushman/Hottentot in whom lumbar lordosis is more marked than in Caucasian, and one wonders whether this may be causally related to the backache.

The epidemiology of backache

In surveys undertaken for the Arthritis and Rheumatism Council in Britain low back pain/leg pain was recorded in 40% of males and 33% of females. Past pain reached a peak incidence at 50 years in both sexes, and it declined thereafter. Extrapolation of this data from Britain to the population of Namaqualand should not be made, but it is of interest to record that in the British surveys disc degeneration, lumbar spondylosis and sacroiliac osteoarthrosis was the cause of backache in 12%, 5% and 2% respectively. It has also been estimated

that disc prolapse probably accounted for less than 10% of backache. These causes of backache account for only one third of the complaints of backache (Lawrence 1969). The lack of lumbar spine X-rays is a deficiency of the Namaqualand study particularly because of frequency of historical data about backache. The male subjects of this survey were or had been labourers and it is well-known that occupation does influence the severity and prevalence of backache from degenerative lumbar disc disease (Kellgren and Lawrence 1952; Lawrence 1955; Lawrence 1969b). It would not be unreasonable to expect a considerable number of the Namaqualand subjects to show evidence of lumbar disc degeneration.

CHAPTER 6

RHEUMATOID ARTHRITIS

Felix qui potuit rerum cognoscere causas
sed fugit interea, fugit in reparabile
tempus. Virgil. 70 - 19 BC.

INFLAMMATORY JOINT DISEASE

1.0. RHEUMATOID ARTHRITIS

The cause of rheumatoid arthritis is unknown but as advances are made it is likely that the unitary concept which is implied by the term rheumatoid arthritis, will have to change. It is currently believed that the disease results from the interaction of an inciting agent or agents with a suitable genetically determined environment. The inciting agent(s) is at present unknown but bacteria, mycoplasmas and viruses have all had their proponents. A good review which summarises the search for an infective agent in rheumatoid arthritis has been given by Marmion (Marmion 1976). The bacteria which have been implicated are Diptheroids (Stewart, Alexander and Duthie 1969) and Clostridium perfringens (Olhagen and Monnson 1968). The rather tenuous evidence for a mycoplasmal infection as the cause for rheumatoid arthritis has also been reviewed exhaustively (Taylor-Robinson and Taylor 1976). At present a viral aetiology remains the most tenable in spite of several constraints (Phillips 1976). Currently, the virus which is regarded as a strong candidate is the Ebstein-Barr virus because of the demonstration that the serum of rheumatoid arthritis patients contains antibodies against a nuclear antigen derived from EB virus infected cells which can be demonstrated either by precipitation or immuno-fluorescent techniques (Alspaugh and Tan 1976; Alspaugh, Jensen and Rabin et al 1978; Vaughan, Catalano, Jensen et al 1978). Another attraction of the Ebstein-Barr

virus is the well-known ability of this virus to infect B lymphocytes and to stimulate them to replicate and produce antibody (Vaughan, Catalano, Jensen et al 1978). One of the major difficulties which has beset all the infective theories of rheumatoid arthritis has been the inability of the putative infective agent to fulfill Koch's postulates. The animal models of arthritis are much more interesting and they offer plausible ideas of how rheumatoid arthritis may arise or be sustained (Hadler 1976). The animal experiments are based on the similarity of the disease to human rheumatoid arthritis and they include such conditions as Erysipelothrix infection of pigs (Drew 1972), the mycoplasmal infections of rats, mice and turkeys (Ross 1973), and adjuvant arthritis in the rat (Pearson 1956). Recently another animal model induced by streptococcal cells or cell walls in rats has raised the possibility of undegradable cell wall products as a source of antigenic persistence (Cromartie, Craddock, Schwab et al 1977). All the animal models however also have deficiencies because they do not exactly mimic the human disease with its characteristic relapses and remissions. The two models which are potentially the most interesting are the rat adjuvant, and the rat streptoccal cell wall arthritis model because these provide a plausible mechanism of antigen persistence and they raise the further possibility that bacterial cell wall products (Peptidoglycans) which are ubiquitous in nature could be one reason for the persistence of rheumatoid arthritis. If bacterial products are the pathogenetic agents it will explain why

there have been so few isolates of viable bacteria from rheumatoid joints. The soil in which the inciting agent acts to produce the disease has until recently received little attention, but with the development of tissue typing techniques and the identification of the D-locus antigens this field is gradually being explored. There is now a strong association between HLA-DRW4 and the familial occurrence of rheumatoid arthritis, (Statsny 1978) while HLA-DRW3 is associated with worse disease and greater susceptibility to the toxic effects of remission inducing drugs (Panayi, Wooley and Batchelor 1978; Batchelor, Wooley, Panayi et al 1979). The interaction between seed and soil produces the inflammatory reaction which is characteristic of the disease. The following is an outline of the current thinking about the pathogenesis of rheumatoid arthritis (Harris 1981):

1. An immune response is triggered in the host by the inciting agent. The major determinant is the genetically controlled immune response which determines the mode and intensity whereby antigen is recognised as well as the degree to which the immune response will be amplified and the antigen localised.

2. Antigen-antibody complexes form in the joint cavity and become trapped in avascular hyaline and fibrocartilage.

3. In the synovial fluid the immune response activates many processes which interact and produce self sustaining active inflammation. The activated systems include complement, kinins, blood clotting and fibrinoysis.

4. Mediators produced by the interaction of mononuclear cells stimulate the synoviocytes to proliferate and to produce proteinases and prostaglandins, and deeper in the synovium fibroblasts are activated to produce more connective tissue matrix. In the synovium the immune response generates the production of rheumatoid factor which following the interaction with IgG causes further inflammatory reactions. (It is believed by some that sufficient IgM-IgG interaction takes place to sustain the inflammatory reaction in the synovium).

5. The synovial response becomes polarised and it spreads to invade the cartilage and sub-chondral bone through the release of proteases and elastases.

6. In order for the progressive destruction of joint tissue to continue the normal mechanisms which inhibit inflammation and degradative enzymes must be overwhelmed and this occurs through saturation of synovial fluid inhibitors and tissue inhibition of specific enzyme system.

1.1.0. The criteria for the diagnosis of Rheumatoid Arthritis

There are no pathognomonic signs of rheumatoid arthritis. The classical association of subcutaneous nodules with the characteristic histological appearance is also not pathognomonic (Moore and Wilkens 1977; Wood and Beerman 1960). The diagnosis therefore depends upon the concurrence of a number of physical signs and laboratory features, which have been grouped together to form the American Rheumatism and Arthritis (ARA) criteria (Ropes, Bennett, Cobb et al 1959).

THE ARA CRITERIA FOR RHEUMATOID ARTHRITIS

1. Morning stiffness
2. Pain on motion or tenderness of at least one joint
3. Swelling (soft tissue/fluid) in at least one joint (observed by a physician)
4. Swelling of at least one other joint observed by a physician
5. Symmetrical joint swelling
6. Subcutaneous nodules
7. X-ray changes typical of rheumatoid arthritis
8. Positive rheumatoid factor (by a method which is not positive in more than 5% of normal controls)
9. Poor mucin precipitate from synovial fluid
10. Characteristic histological changes in the synovial membrane (Three or more of the following - villus hypertrophy, proliferation of synoviocytes, infiltration with lumphocytes/plasma cells, lymphoid aggregation, foci of cell necrosis and deposition of fibrin.
11. Characteristic histological changes in nodules

These criteria have been used extensively in arthritis clinics and in epidemiological studies. In clinics they have been easy to use because it is possible to ensure that the long list of exclusions can be excluded with reasonable certainty. In epidemiological field studies they do not lend themselves as readily because a number of the criteria such as synovial analysis, nodule histology cannot be undertaken easily. Another difficulty has been that the criteria have sometimes failed to detect inactive rheumatoid arthritis. Because of these problems these criteria were replaced by modified criteria which came to be known as the Rome criteria for active and for inactive rheumatoid arthritis (Kellgren, Jeffrey and Ball 1963):

THE ROME CRITERIA

Active Rheumatoid Arthritis	Inactive Rheumatoid Arthritis
1. Morning stiffness	1. Past history of polyarthritis
2. Pain on motion or tenderness of at least one joint (observed by physician)	2. Symmetrical deformity involving at least one hand or a foot
3. Swelling (soft tissue or fluid) of at least one other joint (observed by physician)	3. X-ray changes of rheumatoid arthritis

4. Symmetrical joint involvement

4. Positive rheumatoid factor

5. Subcutaneous nodules

6. X-ray changes of rheumatoid arthritis

7. Positive rheumatoid factor

Classical rheumatoid arthritis = 7-8 criteria Definite = 3-4 criteria
Definite rheumatoid arthritis = 5-6 criteria Probable = 2 criteria
Probable rheumatoid arthritis = 3-4 criteria

These modifications of the criteria were considered to be more practical, but they raised new doubts because considerable discordance arose in less severe cases, and because many of the subjects with multiple clinical criteria in both categories (active or inactive) were considered to have osteoarthrosis by the field workers. Furthermore from the laboratory point of view many of the subjects with positive tests for rheumatoid factor or those with erosions had no clinical evidence of rheumatoid arthritis (Kellgren 1966). Renewed attempts were made in 1966 to resolve these difficulties with a new set of criteria which came to be known as the New York Criteria (Bennett and Wood 1968). These criteria were again subdivided into criteria for active polyarthritis and criteria for rheumatoid arthritis. The criteria are as follows:

THE NEW YORK CRITERIA FOR RHEUMATOID ARTHRITIS

1. A history past or present of an episode of joint pain involving three or more limb joints (Duration not stipulated). Joints on either side count separately and groups of joints such as PIP or MCP joints are counted as a single joint each.

2. Involvement by swelling, limitation of motion, subluxation or ankylosis of at least three limb joints, of which two of the joints must be symmetrically involved, and at least one joint must be a hand, wrist or foot. (Excluded from this criterion are the DIP joints, the fifth PIP, first CMC, hips and first MTP joints).

3. X-ray features if grade 2 or more erosive arthritis (Kellgren, Jeffrey and Ball 1963a).

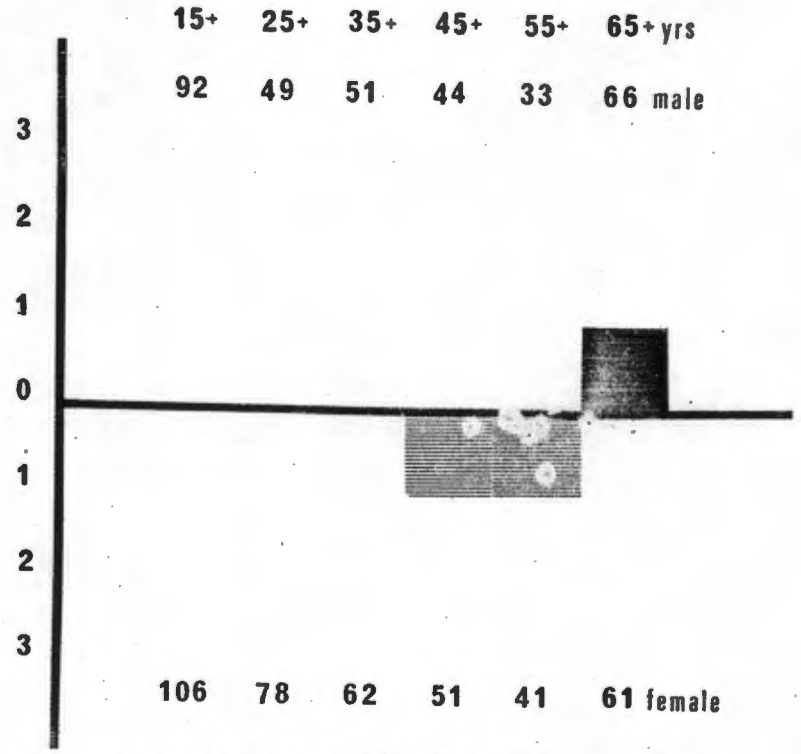
4. A positive serological reaction for rheumatoid factor.

The first two criteria are the essential components and if they are not present, the criteria lose specificity. The second criterion which is concerned with physical signs is the most important. In a comparative study, 93% subjects with 4 - 5 positive ARA criteria met the second New York criterion. Furthermore 2.25% of the subjects meeting the second criterion were seropositive (Allander 1970). In this study it was decided to use the New York criteria because they appear to be the best

set of criteria so far, with the fewest limitations, recognising that aspects of the second criterion do present some difficulty notably in the early stages of a survey where the normal range of motion of joints is not established for the population under study.

1.1.1. Application of the New York Criteria to the Rietpoort population

Three individuals, two females and one male satisfied the first two New York criteria. Their ages were 54, 57 and 72 years. The grading of severity of clinical joint involvement was as follows: Grade 2 = 1, Grade 3 = 2, Grade 4 = 0. All the subjects had evidence of erosive arthritis on X-rays of their hands and feet and all three had positive tests for rheumatoid factor. The positive rheumatoid factor tests were distrubuted as follows; HEAT (SCAT) test = 1, LATEX agglutination test = 3. The LATEX titres were 1000, 1000, 160 in the two females and the one male subject respectively. The prevalence of rheumatoid arthritis is thus 0.40%. The age specific prevalence is shown in Figure 6.1.1.



AGE-SPECIFIC PREVALENCE OF RHEUMATOID ARTHRITIS

Fig. 6.1.1.

The age specific prevalence for females is 1.96% and 2.4% for the 45+ and 55+ age groups, and 1.5% for males in the 65+ age group. Since rheumatoid arthritis occurs more frequently after the age of 35 years, the prevalence was determined for those subjects over 35 and it was found to be 0.73%.

Comparison of Rietpoort and other South African studies

The ARA criteria and the modified criteria used in previous South African studies (Beighton, Solomon and Valkenburg 1975; Solomon, Robin, Valkenburg 1975; Meyers, Daynes, Beighton 1977) were also used to provide a comparison with the New York Criteria. Using the ARA criteria 5 subjects were regarded as definite and probable rheumatoid arthritis giving a prevalence of 0.68%. The modified Rome criteria identified 4 subjects (2 males and 2 females) with definite rheumatoid arthritis and one male with probable rheumatoid arthritis, giving a prevalence of 0.68% for definite and probable rheumatoid arthritis (0.54% definite, 0.13% probable). Three of these subjects also fulfilled the New York Criteria which gives a concordance of 60%. The comparison of the Rietpoort RA prevalence with the other South African studies is shown in the following Table 6.1.1.

TABLE 6.1.1.

THE PREVALENCE OF RHEUMATOID ARTHRITIS IN SOUTH AFRICA (ROME CRITERIA)

RURAL	CLINICAL ARTHRITIS	DEFINITE R.A.	DEFINITE + PROBABLE
TSWANA	0.9%	0.12%	0.87%
XHOSA	1.7%	0.68%	2.20%
COLOURED (Rietpoort)	0.8%	0.54%	0.68%
URBAN			
TSWANA	4.4%	0.90%	3.30%

The prevalence of rheumatoid arthritis in other parts of the world

Studies of the prevalence of rheumatoid arthritis have been undertaken in many parts of the world. In the United Kingdom the prevalence is 1% for adult males, and 3% for adult females, while in Holland it is 0.4% for males and 1% for females (Kellgren, Lawrence and Aitkin-Swan 1953; de Graaf 1959). The largest study to date is that of the U.S. National Health Survey in which the ARA criteria were used where the prevalence was 0.7% in males and 1.6% in females (Engel 1968). When the New York criteria and ARA criteria were compared in the town of Sudbury, 4% females and 1% males were considered to have definite rheumatoid arthritis by the ARA criteria (overall 0.9%), while 0.6% of females and 0.1% of males were positively identified as RA by the New York criteria. The New York Criteria are more stringent in their requirements for diagnosing rheumatoid arthritis, which is useful in field studies. The following Table 6.1.2. provides a comparison of the prevalence of rheumatoid arthritis using the New York Criteria.

TABLE 6.1.2.

A COMPARISON OF PREVALENCE RATES IN DIFFERENT POPULATIONS USING THE CLINICAL COMPONENTS OF THE NY CRITERIA

AREA	SOURCE	MALES	FEMALES
Britain	Lawrence (1977)	1.9%	2.8%
USA (Sudbury)	O'Sullivan (1972)	0.14%	0.6%
USA (Arizona)	Henrard (1975)	6.7%	5.9%
Rietpoort (South Africa)	Meyers (1982)	0.29%	0.5%

There is not much to recommend the New York criteria over the ARA criteria in clinical situations. This is supported by a clinic study which has showed that the ARA criteria have a sensitivity of 90% compared to 93% with the New York criteria and 91% for the Rome criteria (Chalmers, Danchot, Kellgren et al 1970).

Several studies have shown that latitude plays no part in determining the prevalence of rheumatoid arthritis (De Graff, Laine and Lawrence 1963; Bunim, Burch and O'Brien 1964; Gofton, Robinson and Price 1964; Lawrence, Behrend, Bennett et al 1966), but there is some evidence that longitude may play a role in determining the occurrence of rheumatoid arthritis (Sita and Sebo 1968).

Urban/rural differences have not been found to explain the occurrence of rheumatoid arthritis elsewhere (Engel 1968). Several studies have shown that the prevalence of the disease is if anything higher in rural living people (Lawrence 1961; Mikkelsen, Dodge, Duff et al 1962; Al-Rawi, Alazzawi, Alajili et al 1978; Lawrence, Bremner, Ball et al 1966) as compared with urbanised communities (de Graaf 1959; Kellgren 1966; Lawrence 1969a). Other reports suggest that the prevalence is the same for urban/rural groups in Sweden, and that urban living Finns have a higher prevalence than rural Finns (Hellgren 1970). The South African studies are in marked contrast to most of the other published data. In South Africa the prevalence of rheumatoid arthritis is different, being lower in a rural community compared with an urban community. These studies are also unique in the following respects

They are the only studies which have compared the prevalence of rheumatoid arthritis in a defined group of individuals under two different circumstances, and they compare relatively unsophisticated simple life styles (tribal) with urban living. In the South African populations rural has meant living a simple life style in single or small groups of houses, with no amenities which characterise urban living. In the Rietpoort study the population comprised individual families or small numbers of a family living together, often separated from others by some distance, with none of the amenities of a town/city life such as water bourne sewerage, electricity, piped water and other public health facilities and the circumstances were almost identical to other rural S.A. groups. An examination of most of the reported studies does not clarify what is meant by rural and the only reported rural studies which resemble the South African ones, are those amongst the North American Indian populations living in reservations. In these North American Indian populations the prevalence has ranged from 0.7% in the Haida (Gofton, Robinson and Price 1964) to 1.4% in the Blackfeet 3.4% in the Yakima (Beasley, Wilkens, Bennett 1973) and 6.8% in the Chippewa (Harvey, Lotze and Stevens 1981). This last study did not employ the generally accepted epidemiology criteria for rheumatoid arthritis. Another difficulty is that different age groups have been used to calculate the prevalence. In some the prevalence is given for the whole population while in others the prevalence has been calculated for the those over 30 years. These studies and others are summarised in the following Table 6.1.3.

TABLE 6.1.3.

THE COMPARATIVE PREVALENCE OF RHEUMATOID ARTHRITIS IN RURAL/URBAN POPULATIONS

POPULATION	URBAN/RURAL	AGE	DEFINITE R.A. (%)	CRITERIA
ENGLAND				
Leigh/Wensleydale (Lawrence 1961)	Urban/Rural	15+	1.1%	ARA
EUROPE				
Czechoslovakia (Sitaj 1968)	Urban/?Rural	15+	1.0%	ARA
Bulgaria (Tzonchev 1968)	Urban	15+	0.86	ARA
Sweden (Hellgren 1970)	Rural	ns.	0.5%	ARA
Sweden (Hellgren 1970)	Urban	ns.	0.5%	ARA
JAPAN				
Hiroshima/Nagasaki (Wood 1967)	Urban	15+	0.4%	ARA
MIDDLE-EAST				
Israel (Adler 1967)	Urban	20+	2.0%	ARA
Iraq (Al-Rawi (1978)	Urban	16+	1.0%	ARA
NORTH/CENTRAL AMERICA				
Tecumseh (Mikkelsen 1967)	Urban	6+	0.39	ARA
Pittsburgh (Cobb 1957)	Urban	14+	0.7%	NY
Sudbury (O'Sullivan 1972)	Urban	15+	0.9%	ARA/Rome
Jamaica (Lawrence 1966)	Rural	35-64	2.08	ARA
NORTH AMERICAN INDIAN GROUPS				
Eskimo (Beasley 1973)	?	15+	0.8%	ARA
Haida (Gofton 1964)	Rural	15+	0.7%	ARA
Pima (O'Brien 1963)	Rural	30+	1.5%	ARA
Blackfeet (O'Brien 1963	Rural	30+	1.4%	ARA
Chippewa (Harvey 1981)	Rural	18+	6.8%	ARA
Yakima (Beasley 1973)	Rural	18+	3.4%	ARA
SOUTH AFRICA				
Negro (Beighton 1975)	Rural	15+	0.1%	Rome
Negro (Solomon 1975)	Urban	15+	0.9%	Rome

The epidemological investigations which have thus far been undertaken have shown a wide variation of prevalence and no racial group has been shown to be immune. The disease is uncommon in Japanese (Shichikawa 1968) and in Eskimos (Beasley, Rotailla, Healey 1973). The hopes of the rheumatologists and epidemologists of the early sixties that the tool of epidemiology would clarify the causes of rheumatoid arthritis have not been realised and it was partly this that led some to conclude that there is no justification for a major field survey which aims only at determining the point prevalence of rheumatoid arthritis in a particular population group (Cobb 1965), but an alternative view, still expressed, is that such studies do have value in testing hypotheses about rheumatoid arthritis and for planning health care (Allander and Bjelle 1981).

The prevalence of rheumatoid arthritis in Africa

Studies to determine the prevalence of rheumatoid arthritis in Africa have been sparse (Muller 1970) and clinical reports have emphasised the infrequent occurrence of the disease (Hall 1966; Gelfand 1969; Kanyerezi 1969; Greenwood 1969; Anderson 1971; Percy-Lancaster 1974; Blumsohn 1976; Bagg, Hansen, Lewis et al 1979). Epidemiological studies in South Africa has shown that tribalised Blacks have a prevalence of 0.12 - 0.68% of definite rheumatoid arthritis and probable + definite rheumatoid arthritis of 0.87% - 2.20% (Beighton, Solomon, Valkenburg 1975; Meyers, Daynes, Beighton 1979) while in an urbanised Black community of Soweto the prevalence of definite and definite plus probable rheumatoid arthritis was 0.9% and 3.30%

respectively. These findings have raised the question of urbanisation as a factor in the genesis of rheumatoid arthritis. There is no published information about the prevalence of rheumatoid arthritis in Coloured South Africans, and for this reason as well as the unique opportunity of measuring the prevalence of the disease under nearly similar circumstances a start was made in the rural community of Rietpoort. The data also indicates a low prevalence of the disease and preliminary data in an urban Coloured community show that there is a significant difference ($p = 0.05$) in prevalence of rheumatoid arthritis in females over the age of 35 years (Meyers 1980) when compared with the Rietpoort data.

Is rheumatoid arthritis a new disease?

There has been argument about the antiquity of rheumatoid arthritis which has been well discussed (Short 1974). From this and other reports it does seem that the disease has existed for centuries (Caughey 1979), but what is more interesting however is that this disease was not clearly described before the 19th century (Parish 1963), and it was not until 1858 that the term rheumatoid arthritis was introduced to differentiate it from other chronic joint diseases by Sir Archibold Garrod (Garrod 1859). From this time onwards the disease was increasingly reported (Virchow 1858; Charcot 1867; Charcot 1889). This does suggest that there was an increase in the prevalence of rheumatoid arthritis and this increase coincides with the industrialisation/urbanisation of Europe. The great interest of the South African studies is based on their support for a putative

environmental agent which occurs in urban dwellers, and in this way it lends some support to the idea that rheumatoid arthritis is a disease of urbanisation. What the possible effects can be are totally unknown. Some support for these ideas can also be found in an interesting recent publication on arthritis in Saxon and mediaeval skeletons. A radiological examination of the skeletal remains showed erosive arthritis in three skeletons of which one was a possible rheumatoid arthritis (Rogers, Watt, Dieppe 1981).

1.2. EROSIVE ARTHRITIS

Erosive arthritis was found on the hand and foot radiographs in 39 subjects. The relationship to previous history of arthritis and to rheumatoid factor is shown in the following Table 6.2.1.

TABLE 6.2.1.

EROSIVE ARTHRITIS OF HANDS/FEET

JOINT GROUP	NO:	HISTORY OF ARTHRITIS	CLINICAL ARTHRITIS	POSITIVE RHEUMATOID FACTOR
PIP	24	8	3	4
MCP	6	3	3	3
WRIST	1	0	0	0
MTP	8	6	3	3
IP JOINT BIG TOE	3	2	0	0

The subjects with erosive disease of the PIP/MCP/MTP joints who had clinical evidence of arthritis and positive rheumatoid factor were the three who fulfilled the New York criteria for rheumatoid arthritis. It can be seen from the table that of 21 subjects with erosive arthritis of the PIP joints only 5 gave a previous history of arthritis and one an elderly male of 76 years had a positive rheumatoid factor. Three subjects with erosions of the MCP joints also gave no history of previous arthritis and had negative rheumatoid factors. The 5 subjects with erosions of the MTP joints who did not have rheumatoid arthritis gave a history of previous arthritis either in the hands, or large weight-bearing joints. The prevalence of erosive arthritis in this population is 5.58%. Similar findings have been reported in other studies which have also demonstrated a lack of concordance between radiological erosions and clinical or serological evidence of arthritis (Lawrence 1977). The reasons for these erosive changes have been discussed. Direct trauma to a joint can cause an erosion to develop in articular cartilage, (Crock 1964) and erosive arthritis of the feet has been related to walking barefoot and the consequent exposure to trauma. Support for a traumatic basis for some of these erosive changes comes from other studies which have shown that where the predominant occupation in males involved the possibility of micro trauma to the hands, erosions of the hands was greater in those so exposed (Lawrence 1977h). Further support for the role of trauma comes from studies which have shown that erosions of rheumatoid arthritis are greater in the dominant hand, and that paralysis of a limb decreases the erosions in the paralysed limb (Glick 1967).

1.3. RHEUMATOID FACTOR (see chapter 11)

There were 18 subjects who had a positive Latex agglutination test (10 males and 8 females) and 11 subjects with a positive HEAT test (7 males, 4 females). The prevalence of rheumatoid factor in the population was 2.5% with the Latex test and 1.7 for the HEAT test. An examination of the 18 Latex positive subjects showed that 6 gave a history of previous arthritis and 5 complained of morning stiffness in excess of 15 minutes in the past. Three of these subjects were suffering from rheumatoid arthritis and one was considered to have probable rheumatoid arthritis. If the 3 subjects with definite and the 1 subject with probable rheumatoid arthritis were excluded the 5 subjects with morning stiffness and 2 with previous arthritis which can be compared with 8 subjects with positive Latex tests with no history of rheumatic complaints. There was thus no association between rheumatic complaints and a positive rheumatoid factor.

2.0. JUVENILE CHRONIC ARTHRITIS

Two subjects, a male of 62 and a male of 16, were considered to have juvenile chronic arthritis. The older male gave a history of a febrile illness in childhood which left him with contractures of his knees. At the time of the survey there were marked contractures of the knees and reduced motion of the right elbow. X-rays showed no evidence of joint erosions, and the rheumatoid factor was negative. The younger male complained of morning stiffness, backache and joint pains for longer than 3 months. Examination showed arthritis of the MCP, PIP and MTP joints. The rheumatoid factor was negative, and

there were no other obvious diseases to account for his illness. Both these individuals meet the criteria for juvenile 'rheumatoid' arthritis proposed by the symposium on Population Studies of the rheumatic diseases (Bennett and Wood 1968). The prevalence of Juvenile Chronic arthritis amongst children in this survey is therefore 0.18%.

3.0. PSORIATIC ARTHROPATHY

6 subjects had psoriasis. In 5 there was evidence of active psoriasis, and in one there was a previous history of psoriasis. The mean age of these subjects was 43 years, three were males and three females. The prevalence for the population was 0.81%. None of these subjects had a past history of arthritis and there was no clinical evidence of arthritis. Psoriatic arthropathy occurs in patients with psoriasis in 0.4% to 49.0% cases. The Cape Town experience of psoriatic arthropathy is that 15% of psoriatic patients will exhibit some form of peripheral arthritis while 41% will show radiological evidence of sacroiliitis (Green, Meyers, Gordon et al 1981). There are few reported attempts to measure the prevalence of psoriatic arthropathy in the field. One such attempt at measurement, estimated the prevalence as 5/100,000 (Lumhold 1963) but there are no generally acceptable criteria and it seems that this is not a diagnosis which will easily be made in an epidemological field study such as the one in Namaqualand, given the small number of subjects with psoriasis. One must agree with Lawrence who has suggested that if criteria are defined and strictly applied the diagnosis will be so rare that it will seldom be made in population

samples (Lawrence 1977i). If the Cape Town data on psoriatic arthropathy is extrapolated it would require a prevalence of psoriasis of at least 10% to yield any possibility of finding psoriatic arthropathy.

4.0. ANKYLOSING SPONDYLITIS

No subjects were found with clinical ankylosing spondylitis. It was not possible to take X-rays of the sacro-iliac joints so that it is likely that subclinical or mild disease was missed. Part of the questionnaire which the respondents completed about backache was directed at historical features which suggest the presence of inflammatory backache, because of the recent suggestions that the clinical history is a sensitive and specific indication of ankylosing spondylitis (Calin, Porta, Fries et al 1977). It is probably more correct to say that the historical features are predictive rather than specific for ankylosing spondylitis. Eight males and thirteen females gave a backache history which fulfilled these historical criteria. This gave a prevalence of 4.8% of males and females under the age of 45 years (4.2% for males and 5.3% of females). None of these twentyone subjects showed clinical evidence to suggest ankylosing spondylitis. For this study the clinical components of the criteria for ankylosing spondylitis were used. They are limitation of motion of the lumbar spine in all three planes - (anterior flexion, lateral flexion, extension) a history of a presence of pain in the lumbar spine. Limitation of chest expansion to 2.5 cm. or less at the level of the 4th intercostal space, was not used because

no information on the range of chest expansion for this population was available. The lack of X-rays is however a deficiency of this study. It is also impossible to predict the possible numbers of affected individuals because the HLA tissue types of the community are not known. In Caucasians it has been predicted that 20% of those who are B27 are at risk from developing ankylosing spondylitis. In Coloured subjects the prevalence of B27 is 4.8%. If this is applied to this community it is possible to suggest that of the 734 adults, 35 could have had the genetic endowment of B27 and 7 would be at risk for developing ankylosing spondylitis. This is a theoretical estimate and it depends on an assumption that B27 is the same in the Rietpoort community as it is in Coloured subjects from Cape Town.

5.0. CRYSTAL ARTHRITIS

Clinical gout was not seen. The uric acid profile of this community is discussed elsewhere (Chapter 11). Three males aged 18, 37 and 48 showed calcification in the triangular cartilage at the wrist. This was asymptomatic in 2 and in the third there was a history of recurrent inflammatory episodes in the hand. No further X-rays are available from these subjects to establish whether this was a generalised chondrocalcinosis, but calcification of the triangular cartilage is so characteristic of chondrocalcinosis that it seems inescapable that these subjects did have this condition and this is particularly so for the one subject with positive history (McCarty 1975). There were four individuals whose hand radiographs

suggested hydroxyapatite crystal deposition disease (HADD). None were symptomatic and their ages were 57 years, 65 years and 75 years for the males respectively and 57 years for the females.

6.0. SYSTEMIC LUPUS ERYTHEMATOSUS

No subjects were found to have systemic lupus erythematosus (see chapter 13 for a discussion of antinuclear antibody). There were 2 subjects with chronic discoid lupus which gives a prevalence of 0.27% for adults over the age of 15 years.

7.0. UNEXPLAINED ARTHRITIS

7.0.1. Active arthritis/tenosynovitis

Five subjects were found to have an active synovitis. The mean age was 63.6 years. In one male age 72 years there was a dorsal tenosynovitis of the right wrist. This man also had a reduced range of motion of the elbows, ankles and metatarsophalangeal joints; there was no preceeding history of arthritis and the radiographs showed mild osteoarthrosis of the hands. Three subjects had a subacute arthritis of the left elbow (male 75 years), left knee (male 72 years) and right ankle (female 61). The male with the elbow arthritis gave no preceeding history of arthritis and the radiographs were unhelpful. The two other patients had generalised osteoarthritis. A male of 38 years had swelling of the left big toe joint. There was no preceeding history of acute arthritis in the joint but he did complain of previous episodes of knee arthritis. The uric acid level was 0.13 m.mol/L., and the radiographs were unhelpful. The cause of these instances of subacute arthritis is

uncertain in some of these subjects, while in those with well-marked osteoarthrosis this process may be invoked as a cause. Inflammatory episodes in osteo-arthritis are well-known (Kellgren and Moore 1952; Erlich 1972), and they may be provoked either by crystals of calcium pyrophosphate (McCarty 1975) of calcium hydroxyapatite (Dieppe, Huskisson, Crocker et al 1976). In others where no cause is found, trauma may play some pathogenic role. In the one male with arthritis of the big toe, gout seems likely but there is little to support this. There were also no radiographic features to suggest chondrocalcinosis as a cause.

7.0.2. Tender joints

There were three subjects (two males and one female) aged 76 years, 75 years and 73 years respectively who had tenderness of the MTP joints for which no cause was found. One male and the female had no previous history of arthritis while the second male had had a previous arthritis lasting for 7 days.

7.0.3. Decreased range of motion of joints

There were eleven subjects in whom there was a reduced range of motion of one or several joints or groups of joints (shoulder measurement is discussed in chapter 8). All but one of these subjects were males, and the average age was 76.6 years. Very few gave a history of previous arthritis and in 4 subjects the restriction in the range of joint motion was found in several joint groups. Radiological osteoarthritis of the hands was not a

marked feature of this group. It was mostly confined to the terminal interphalangeal joints and was usually Grade 2. In two the grading was Grade 3 and there was a more generalised distribution of the osteoarthrosis. It is unlikely that previous inflammatory joint disease can explain the reduction of joint range. In the absence of an obvious cause it can be assumed that these subjects represent either an extreme of joint range for the population, or a 'natural' decrease of joint mobility associated with increasing age. The decreased range of motion of joints is shown in the following Table 6.7.1.

TABLE 6.7.1.

SUBJECTS WITH REDUCED RANGE OF MOTION OF JOINTS

JOINT	NO	MALES	FEMALES	PREVIOUS HISTORY OF ARTHRITIS
PIP	3	3	0	1
MCP	2	2	0	1
WRIST	2	2	0	1
ELBOW	1	1	0	0
KNEE	1	1	0	0
ANKLE	1	1	0	0
MTP	3	2	1	2
TOES	1	1	0	0
GENERALISED	1	1	0	0

The decreased range of motion is probably due to one or more causes such as previous unrecalled joint disease, disuse and the progressive stiffening of the tissues with age (Ridge and Wright 1966).

CHAPTER 7

OSTEOARTHROSIS

There is in the declining years unequal reparation ..... the drier and more porous parts as membranes, tunicles, sinews, arteries, veins, bones, cartilages, most of the bowels are hardly reparable to their loss.

Francis Bacon. 1685.

OSTEOARTHROSIS

Semantic problems

There is still controversy over the terminology of this group of disorders. The term osteoarthrosis has been suggested because of the absence of signs of clinical inflammation of affected joints. However there are inflammatory episodes which can be recognised clinically in the terminal interphalangeal joints and proximal interphalangeal joints and the term inflammatory osteoarthritis has been used to designate this subset. Episodes of inflammation also occur from time to time which may be confirmed by finding raised cell counts in the synovial fluid. At a microscopic level there is ample evidence of chronic inflammation, and microfractures with repair, so that the arguments about the name are perhaps over-emphasised. Furthermore it must be recognised that osteoarthrosis may be the common end result of several pathogenetic pathways some of which are clearly inflammatory (Erlich 1972) while others clearly are not.

The pathology of osteoarthrosis

The pathology follows a variety of patterns of joint failure, and the essential features are deformations associated with deterioration and mechanical loss of articular cartilage and disturbance of the configuration of the joint related to a series of reparative phenomena (Sokoloff 1980). The phenomenon has been recognised in ancient remains of reptilian and mammalian species. The early clinical descriptions recognised its progressive nature and low mortality

(Haygarth 1805), while one description suggests the inability of cartilage to repair itself as a cause (Bacon 1685).

(i) Degeneration of the cartilaginous surface

The cartilaginous surface of the joint develops focal progressive disintegrations. These start as focal areas of softening at points of impact (Radin, Parker, Pugh et al 1973). These areas become fissured and the edges of fissures show an irregular fibrillation. The fissures eventually extend through the tide-mark to the subchondral bone. In the process of fissuring flakes of cartilage are broken off once the tide-mark has been reached and this helps to denude the end of the bone of its cartilaginous surface (Hough, Banfield, Mottram et al 1974). The microscopic appearances are those of decreased matrix proteoglycans, exposure of collagen fibres and an increase of the metabolic activity of chondrocytes. At a biochemical level the water content of the cartilage is increased (Mankin and Thrasher 1975). The chondrocytes show a marked increase of radiosulphate incorporation into proteoglycans, and amino acid (serine, threonine, proline, lysine) incorporation into collagen. Several enzymes notably hyaluronidase cathepsin D, proteases and lyosomal enzymes which are released from the chondrocytes appear in the matrix and the joint fluid (Mankin 1974).

(ii) Changes in the subchondral bone

The major changes in the subchondral bone are eburnation (Harrison,

Schajowicz and Trueta 1953) the formation of subchondral pseudo cysts or geodes and the formation of osteophytes. The bone geodes are globular or pyrriform defects, which may arise in several ways:

(a) Necrosis and microfractures of the bone and adjacent marrow. The microfractures which are a constant feature of severe osteo-arthrosis occur in areas beneath eburnated bone. Geodes form in these areas where several microfractures coalesce.

(b) Raised intra-articular pressure may force synovial fluid down the cartilage fissures into the subchondral bone.

(c) Osteoclastic resorption of bone.

(d) Localised osteoporosis (uncertain).

The development of the geodes presages the collapse of the structural integrity of the bone particularly in the weight-bearing joints. The third change in the subchondral bone is the growth of osteophytes which are a characteristic feature of the disease. They develop very early and in animal models they develop contemporaneously with the earliest biochemical changes and well in advance of histological changes in the articular cartilage (Gilbertson 1975; Marshall and

Olsson 1971). Osteophytes form at the edge of the joint or at tendinous insertions and they expand in a direction opposite to the direction of motion of the joint, but in the course of time they may protrude into the joint cavity where they cause mechanical interference with joint motion. The way in which this series of pathological events develops is unknown because the earliest stages of the disease cannot be accurately determined, and they would in any event be subclinical.

The present concept is that the earliest changes are an increase in the water content of cartilage and a loss of proteoglycan and a change of the glycosaminoglycan components. This is based on animal experiments such as the models of canine cruciate ligament section (Muir 1977), and rabbit knee joint menisectomy (Moskowitz 1973). Osteoarthritic cartilage breakdown can follow a variety of predisposing causes. Circumstances which may alter the resistance against cartilage breakdown:

(1) Where the cartilage has minimal properties as a material but is subjected to an abnormal biomechanical environment for example an inherited or acquired anatomical abnormality.

(2) Where the mechanical environment is normal but the cartilage has an unbeated or acquired deficiency in its wear remittance as a material for example in ochronosis, crystal depositions.

(3) Where day to day wear of apparently normal cartilage has been sufficiently prolonged to give osteoarthritic bone exposure in older subjects (Meachim 1980).

Clinically it is useful to classify osteoarthritis into primary and secondary forms and according to the joint or joint groups involved (Radin, Parker and Paul 1971; Kellgren and Moore 1952) although it must be recognised that the use of the term primary is really an expression of ignorance about causation. Argument still rages about the concepts of the aetiology of primary osteoarthritis which resolves itself into the following. One view holds that the phenomena are due to wear and tear, while the second view suggests that it is an inevitable concomitant of ageing. In reality both are important and this was emphasised by earlier authors who saw osteoarthrosis as the result of an increased susceptibility of articular cartilage to 'wear and tear' as consequence senescent deterioration. (Bennett, Waine and Bauer 1942).

The diagnosis and evaluation of osteoarthrosis

The diagnosis of osteoarthritis presents few problems being based on the clinical features of pain, inactivity, stiffness, deformity and a characteristic radiographic appearance. The prevalence of osteoarthrosis has been studied in several ways; clinically, radiologically and a combination of both. For ease most investigators have used a radiological evaluation to obtain a more comprehensive picture of the prevalence of the disease. The radiographs of the joints are graded in 4 grades of severity:

Grade 1 = a minute osteophyte of doubtful significance

Grade 2 = a definite osteophyte is present with preservation of the joint space

Grade 3 osteophytes with moderate diminution of the joint space

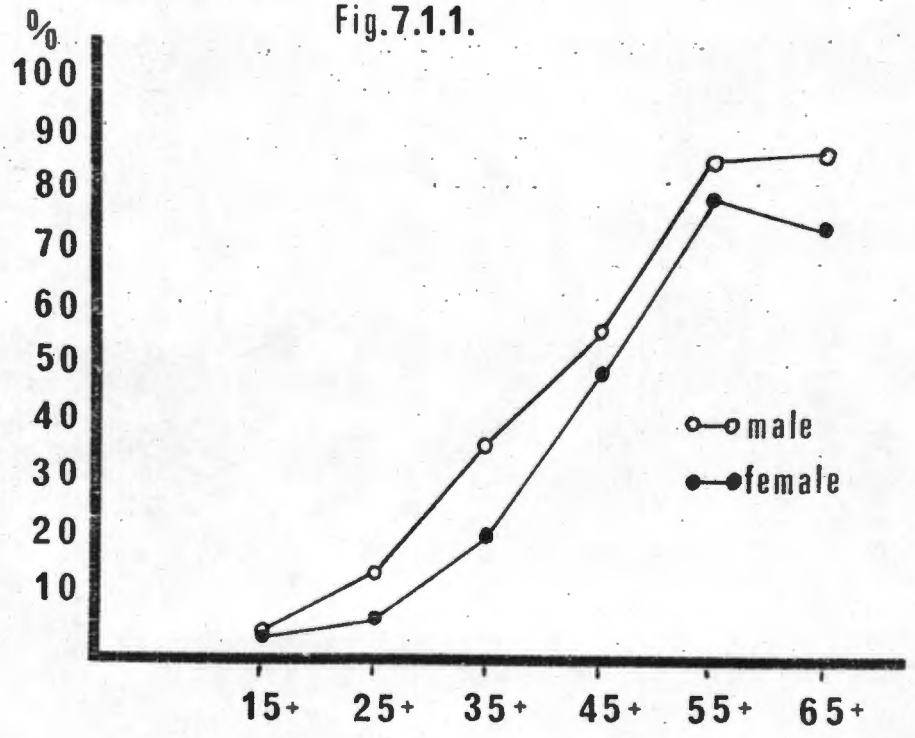
Grade 4 = marked impairment of the joint space with sclerosis of subchondral bone

These radiological criteria give a good measure of agreement between observers (Kellgren and Lawrence 1957) and provide the only reliable method for comparative studies of osteoarthritis (Laine 1968). There has however been some argument about the acceptance of osteophytes alone as evidence of osteoarthrosis.

The prevalence of osteoarthrosis in Rietpoort

In the Namaqualand study no systematic attempt was made to determine the prevalence of osteoarthritis clinically, but Heberden's nodes were noted, and in order to keep costs down, only hand and foot radiographs were taken. It was a specific recommendation of the third international symposium that Heberden's nodes should be included in population surveys because of their association with generalised osteoarthrosis (Kellgren and Moore 1952). The age specific prevalence of Grade 2 - 4 osteoarthrosis is summarised in the following Figure 7.1.1.

FIGURE 7.1.1.

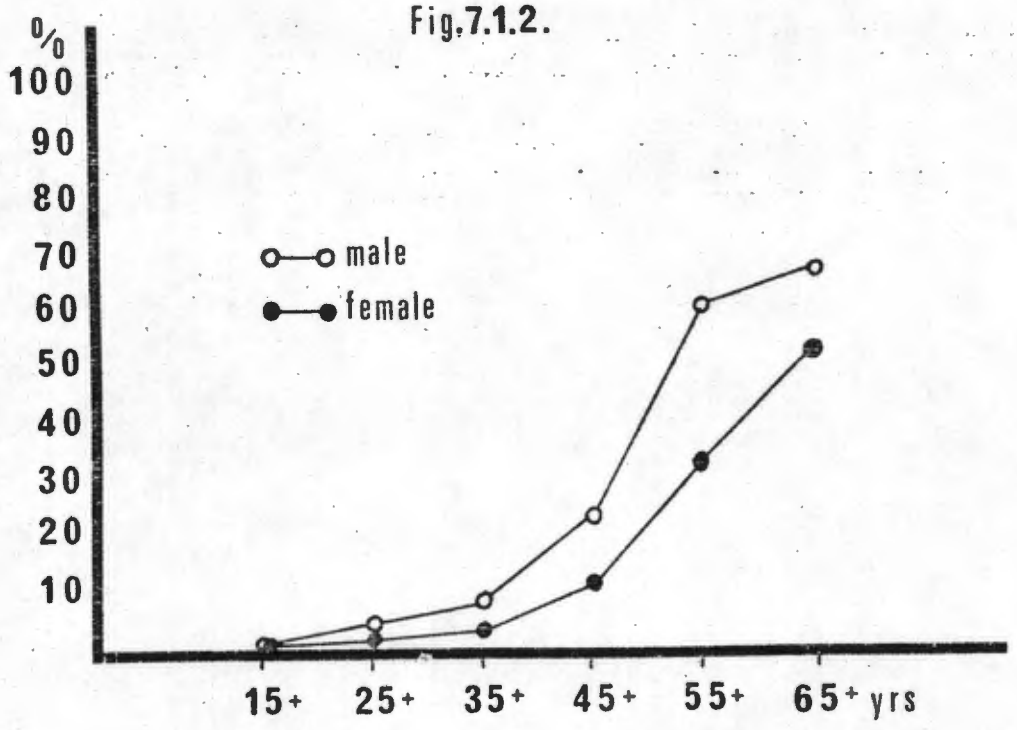


AGE-SPECIFIC PREVALENCE OF OSTEOARTHROSIS GRADE 2-4

The age specific data for grade 2-4 osteoarthrosis shows that th is more osteoarthrosis in males than females up to the age of 45-54 years whereafter the prevalence is nearly the same. By the 65+ decade 90% of males and 75% of females showed some evidence of osteo-arthrosis. The major contribution was made by joints with Grade 2 osteoarthrosis, especially of the metatarso-phalangeal joint of the big toe. In order to assess the severity of osteoarthrosis grade 3-4 osteoarthrosis was assessed separately in the following Figure 7.1.2.

FIGURE 7.1.2.

Fig.7.1.2.



AGE-SPECIFIC PREVALENCE OF OSTEOARTHROSIS
GRADES 3+4

This shows that severe osteoarthritis is present in approximately 10% of the males and the females up to the 35+ decade whereafter the curve for both sexes begins to rise steeply so that in the 65+ decade 70% of men and 55% of women show severe osteoarthrosis. These age specific curves are very similar to those from America and England (Mikkelsen, Dodge and Duff 1970; Gordon 1968; Bennett and Burch 1968; Lawrence 1977f).

The prevalence of osteoarthrosis in joint groups is shown in Table 7.1.2.

TABLE 7.1.2.

THE PREVALENCE OF RADIOLOGICAL OSTEOARTHROSIS IN MALES AND FEMALES

	DIP		PIP		MCP		CMC		1ST MTP	
	MALE	FEMALE	MALE	FEMALE	MALE	FEMALE	MALE	FEMALE	MALE	FEMALE
ALL AGES	18.9	14.5	16.4	12.9	11.9	8.2	9.0	8.2	26.4	26.8
35 YRS +	29.1	22.6	25.3	21.4	18.1	13.2	12.2	13.7	27.2	29.2
35 YRS + (GRADE 3-4)	12.2	7.4	11.6	6.8	13.8	5.1	3.1	3.8	0.62	1.2

An examination of this data shows that osteoarthrosis was more common in males for each joint group except at the 1st MTP joint of the big toe where the difference was reversed. The prevalence of severe disease (Grade 3-4) shows a slightly different pattern of joint involvement in the males. In these subjects the MCP joints were the most frequently involved followed by the TIP and PIP joints. In females the frequency of joint involvement remains unchanged regardless of severity of the process.

The metatarsophalangeal joint of the big toe was the most frequently involved joint but the contribution it makes is due to mild (Grade 2) osteoarthrosis. The next joints in order were the DIP, PIP, MCP and CMC joints. It is a clinical impression that hallux rigidus (OA of the 1st MTP joint) is more commin in males, but this study shows that

this is not so in the Namaqualand community (note that severe OA of this joint was half as common in males as in females 0.62% vs 1.2%).

It is also of interest to determine the number of joint groups affected by osteoarthrosis, and this can be examined as a function of gender and of age. The former attempts to measure the number of joint groups involved in the whole population, while the latter is really an age specific determination.
Table 7.1.3. shows the number of groups of joints involved in the males and females.

TABLE 7.1.3.

NUMBER OF GROUPS OF JOINTS INVOLVED AS A FUNCTION OF GENDER

		NO: OF GROUPS INVOLVED					
SEX	NO:	0	1	2	3	4	5
MALES	252	25%	32%	14.3%	12.3%	11.5%	4.0%
FEMALES	295	30.8%	36%	10.2%	11.5%	8.5%	3.1%

It can be seen from this data that the number of joint groups involved is virtually the same in males and females and that generalised osteoarthrosis (5 groups) forms 4.0% of the male OA and 3.1% of the female osteoarthrosis.
It is more meaningful to look at the data in terms of age because of the increasing frequency of osteoarthrosis with age. This information is presented on Table 7.1.4.

TABLE 7.1.4.

NUMBER OF JOINT GROUPS INVOLVED AS A FUNCTION OF AGE(%)

			NO OF JOINT GROUPS					
AGE	SEX	NO:	0	1	2	3	4	5
15+	M	51	27.5	47.1	5.9	0	0	0
	F	65	53.8	40.0	3.1	0	0	0
25+	M	45	35.6	57.8	4.4	2.2	0	0
	F	62	38.7	54.8	4.8	1.6	1.5	1.5
35+	M	35	8.5	40.0	34.2	8.6	5.7	2.9
	F	52	32.7	48.0	11.5	5.8	2.0	0
45+	M	36	27.8	25.0	25.0	11.1	11.1	0
	F	43	9.3	32.5	11.6	18.6	13.9	0
55+	M	24	8.3	12.4	16.6	29.2	33.3	0
	F	31	9.7	12.9	16.1	38.7	12.9	9.7
65+	M	61	6.6	11.5	16.4	26.2	24.5	14.8
	F	42	14.3	7.1	11.9	23.8	30.9	11.9

The analysis of the number of groups of joints involved as a function of age shows that in the younger individuals large numbers either had no osteoarthrosis, or at most 1 - 2 joint groups involved. As the population ages there are increasing numbers with 3 - 4 joint involvement, and in those over 65 years, 5 joint group involvement occurs in 14.8% of the males and 11.9% of the females. In the females this occurred earlier than in the males.

The osteoarthrotic changes were also examined on a joint by joint basis

(Appendix Table 7A, 7B). In both males and females there was more osteoarthrosis on the right side, and when the individual joints were ranked the following pattern of involvement was found and this is shown in the following Table 7.1.5.

TABLE 7.1.5.

RANKING OF INDIVIDUAL HAND JOINTS

	MALES		FEMALES	
RANK	GRADE 2 - 3	GRADE 3 - 4	GRADE 2 - 4	GRADE 3 - 4
1	DIP 4	MCP 1	PIP 1	PIP 1
2	PIP 1	DIP 2	DIP 4	MCP 5
3	DIP 2	PIP 1	DIP 2	DIP 1
4	DIP 1	MCP2/MCP3	DIP 1	DIP 2
5	MCP 1	-	PIP 5	DIP3/PIP4
6	DIP 3	DIP 5	PIP 3	-
7	PIP 3	DIP 1	DIP 3	PIP5/MCP1
8	PIP 5	DIP 3	PIP 4	-
9	MCP 2	PIP 3	MCP 1	PIP 3
10	MCP 3	PIP 4/5	MCP 2	MCP 2

When the population over the age of 35 years was considered separately no change of this ranking was found, but when the ranking to was done using grade 3 - 4 osteoarthritis a markedly different pattern of involvement was found. In this ranking the involvement of the metacarpophlanageal joints is accentuated in males whereas the

ranking for females remains much the same whatever the grading. If the two hands are considered spearately the ranking order for joint involvement is also different and this is shown in the following Table 7.1.6.

TABLE 7.1.6.

RANKING OF INDIVIDUAL JOINTS FOR RIGHT AND LEFT HAND

MALES	RIGHT	LEFT	FEMALES	RIGHT	LEFT
1	MCP1/MCP3	MCP1		PIP5	PIP5
2	-	DIP2		DIP4	DIP4
3	DIP2	MCP2		DIP1/TIP3	DIP1,2/PIP3
4	PIP1	PIP1		-	-
5	DIP3/MCP2	DIP4		TIP2/PIP5/MCP1	-
6	-	DIP1		-	PIP5
7	DIP1/DIP4	MCP3		-	MCP3
8	-	PIP3		PIP4/MCP1	DIP3/PIP5
9	PIP3	PIP4		-	MCP1/MCP2
10	PIP4	PIP2/DIP3		MCP3	-
11	PIP2/MCP4				

Heberden's nodes were noted in 73 of the females (18.29%) and 71 of the males (21.19%). The age specific prevalence for Heberden's nodes is shown in Table 7.1.7.

TABLE 7.1.7.

AGE SPECIFIC PREVALENCE OF HEBERDEN'S NODES

AGE	MALES	FEMALES
15+	3.3%	6.6%
25+	10.2%	7.7%
35+	15.7%	17.7%
45+	25.0%	21.6%
55+	36.4%	39.0%
65+	56.1%	47.5%

Heberden's nodes were associated with 5 joint arthrosis in 13 subjects, with 4 joint disease in 28 and with 3 joint disease in 36 subjects. In 36 subjects only 1 - 2 joint groups were affected, and in 12 subjects there was no radiological evidence of osteoarthrosis.

Metacarpophalangeal joint osteoarthrosis

An examination of other punlished studies shows that grade 2 - 4 osteoarthritis varies from 5 - 42% but when the more severe grades are considered separately the prevalence is considerably lower and ranges from 0.6 - 17% for males and 0 - 4.0% for females (Lawrence and Sebo 1980). The pooled data from all published surveys show a mean of 13% for males and 14% for females for all grades of osteoarthrosis at the MCP joints, and 3% and 1% for severe OA. A prevalence of 18.1% for MCP OA in the Namaqualand study is not particularly at variance with other reports but a prevalence of 13.8% for grade 3 - 4 osteoarthrosis for males is remarkably high when compared with other studies (Lawrence and Sebo 1980).

An examination of the frequency of individual MCP joint involvement in the Namaqualand subjects shows that it is the 1st - 3rd MCP joints which bear the brunt of the disease and there was a gradient of involvement from the radial side. This was most marked in the males and less obvious in the females. Others have also shown a gradient of severe involvement in males from the radial side for the MCP joints (O'Brien, Clemett and Acheson 1968).

TABLE 7.1.8.

FREQUENCY OF INVOLVEMENT OF MCP JOINTS BY OA (GRADE 2-4-)

	1st	2nd	3rd	4th	5th
MALES	82	55	53	20	6
FEMALES	33	26	30	6	8

The development of severe OA in the 1 - 3rd MCP joints seems to occur from 35+ years in males and increases with each succeeding decades as can be seen from the following Table 7.1.9.

TABLE 7.1.9.

AGE SPECIFIC PREVALENCE OF RADIOLOGICAL OA - GRADE 3 IN THE METACARPOPHALANGEAL JOINTS OF BOTH HANDS (MALES)

	1st		2nd		3rd		4th		5th	
	R	L	R	L	R	L	R	L	R	L
15+	0.00	0.00	1.9	1.9	0.0	0.0	0.0	0.0	0.0	0.0
25+	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
35+	4.3	0.0	0.0	0.0	0.0	0.0	2.1	0.0	0.0	0.0
45+	6.8	0.0	6.8	3.4	6.6	3.4	0.0	0.0	0.0	0.0
55+	20.8	16.6	4.1	8.3	16.6	8.3	0.0	0.0	0.0	0.0
65+	19.6	19.6	8.1	9.8	18.0	0.0	3.2	1.6	0.0	0.0

Similar but less frequent involvement was found in females.

The involvement of the 3rd MCP on the right was particularly striking. There were 26 males and 8 females where this joint was either involved alone or together with the 1st and 2nd MCP joint (Figure 7.1.3. - 7.1.6.). The mean age of these subjects was 67 and 61 years for males and females respectively. None of the subjects recalled trauma to these joints but on 4 of the radiographs there was evidence of previous trauma to the metacarpals and the MCP joints. Most of the subjects were asymptomatic. These rather striking changes require an explanation. The possibility arises of previous inflammatory joint disease but the almost uniform lack of symptoms suggesting inflammatory joint disease in these subjects makes this unlikely. It would also be unusual if this was due to preceeding RA to have affected so many more males than females, and finally the radiological appearances are really not those which are usually seen in RA. There was no evidence or urate of calcium pyrophosphate deposition to account for these appearances. None of these subjects was overtly diabetic and there was no evidence of haemochromatosis in any of 25 subjects in whom the serum iron was measured. One is left with the conclusion that this is may be related to occupation. All the affected males were labourers on surrounding farms and it is possible because of their occupation there may be unusual impact loads distributed over the metacarpal heads leading to osteoarthrosis. It is difficult to see why other joints of the hand do not seem to have developed the same severe changes, but it is presumed that the length of the third metacarpal may influence it by exposing the metacarpal head to more trauma. A similarly high prevalence of MCP OA has been described from Arizona where occupational factors were considered important (Lawrence and Sebo 1980).

MCP OSTEOARTHROSIS (GRADE 4 RIGHT, GRADE 3 LEFT) THE MIDDLE FINGER MCP IS THE WORST AFFECTED

MCP OSTEOARTHROSIS SEVERE DISEASE (GRADE 4)

MCP OSTEOARTHROSIS : GRADE 3 ON THE RIGHT/GRADE 2 ON THE LEFT

MCP OSTEOARTHROSIS : GRADE 3 (NOTE THE RELATIVE SPARING OF THE THUMB)

NON-ARTICULAR/SOFT TISSUE RHEUMATIC DISEASE

1.0. SHOULDER PROBLEMS

Pain in the shoulder is a common clinical problem in general and rheumatological practice. Shoulder problems have not been looked at in any large scale epidemiology study but because of its importance it was decided to attempt to apply epidemiological methods to such a study. For this purpose a question on previous or present shoulder pain was included in the questionnaire. This was used to gauge what the prevalence of shoulder problems are in the community. Secondly a physical examination was undertaken during which the range of motion in abduction, flexion, extension, internal and external rotation were measured. Pari passu with this a series of movements were studied by the following general scheme (Cyriax 1969, Bland, Merritt and Boushey 1979). This scheme involves the systematic examination of the following:

1. Active elevation of the arm, noting the presence of a painful arc.
2. Passive elevation of the arm.
3. Passive scapulohumeral abduction.
4. Passive lateral rotation.
5. Passive medial rotation.
6. Resisted abduction.
7. Resisted medial rotation.
8. Resisted lateral rotation.
9. Resisted adduction.
10. Resisted flexion/extension at the elbow.

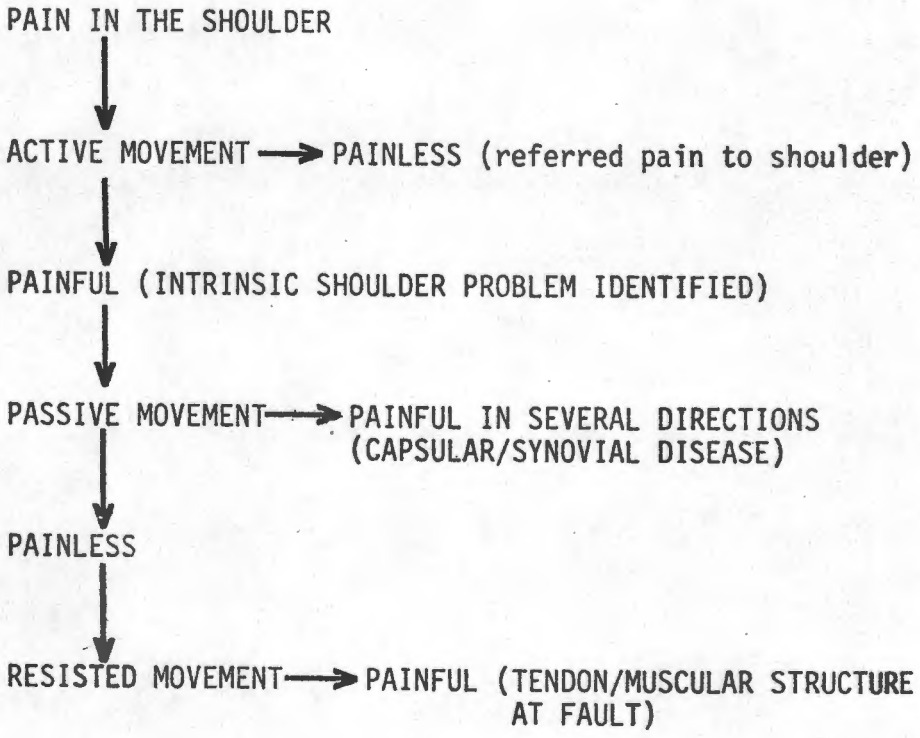
CHAPTER 8

SOFT TISSUE RHEUMATIC DISORDERS

'For age with stealing steps hath clawed me with his clutch'.

Thomas Lord Vaux 1510-1556.

All the passive and resisted movements were measured with the arm at the side. In a few subjects where there was doubt, the rotational movements were measured with the shoulder abducted to the horizontal. Following this series of manoeuvres the pattern of shoulder involvement could generally be resolved by the following algorithm:



This algorithm has been useful in the clinic situation and its utility lies in the sequential application of active, passive and resisted movement. The limitations of the scheme are that degenerative lesions in the shoulder are often multiple so that confusion may arise when there are abnormalities at more than one point of the algorithm. Its use in the field caused neither difficulty nor delays in obtaining all the required information.

Shoulder pain in the population of Rietpoort

The question concerning shoulder pain was 'have you ever had shoulder pain, or do you have pain in your shoulder now?' The response was a simple yes or no. No attempt was made to elaborate on this further unless additional information such as an injury or a dislocation was volunteered. In the following table the numbers of persons responding positively to this question are shown:

TABLE 8.1.1.

Numbers of subjects who have or have had shoulder pain

	15+	25+	35+	45+	55+	65+
MALES	3(3.3%)	14(28.6%)	20(39.2%)	27(61.2%)	20(61.0%)	48(72.2%)
FEMALES	16(15.1%)	32(41.0%)	34(50.0%)	32(62.7%)	19(46.3%)	40(66.1%)
TOTAL	19(9.6%)	46(36.5%)	54(45.4%)	59(62.1%)	39(52.7%)	88(69.29%)

Three hundred and one of the 734 adults in the survey recalled previous shoulder pain or had pain in the shoulder at the time of the study. This gives an overall prevalence of 41.0%. Females exceeded males up to the 45+ decade whereafter the males predominated, and in both there was an increasing number with shoulder pain with increasing age. Others have reported more females with shoulder pain and with a peak at 50 - 60 for males and 60 - 70 for females (Sheldon 1972). Time limitations did not allow an elaborate historical account of shoulder pain so that it must be assumed that most of the shoulder pain had its origins in reversible intrinsic shoulder disease, while in the older age groups referred pain particularly from the cervical spine could also have accounted for some of the shoulder pain. Finally it cannot be

expected that a lay public will accurately define the site of pain without direct questioning, therefore other sources for shoulder pain can also have been the fibromyalgic syndromes around the shoulder girdle and acromio-clavicular arthritis.

Range of movement of the shoulder in the Rietpoort population

The movements which can occur at the shoulder are well known and their range has been agreed on by most authorities (Cyriax 1969, American Academy of Orthopaedic Surgeons, 1965). There is argument about the position of the arm for the measurement of the range of motion and at least one report suggests that the range of external rotation is influenced by the position of the arm (Post 1978). I used the arm at the side method because personal experience supports those who hold that external rotation is not materially affected by the position of the arm. In this population the range of motion of the shoulder was recorded in all subjects but only the data of those in whom there was no history of previous shoulder pain was analysed. The range of motion of flexion was 0^{o} - 90^{o}, of extension 0^{o} - 80^{o}, of internal rotation 0^{o} - 90^{o}, of glenohumeral abduction 0^{o} - 90^{o}, and of external rotation 0^{o} - 90^{o}. The only movement which was limited by age was external rotation which decreased progressively with increasing age. These ranges and the decrease of external rotation are in agreement with other published reports (De Palma 1973). The percentage decrease of external rotation with age is shown in the following figure and it is calculated as a percentage decrease from 90^{o}: (Figure 8.1.1.)

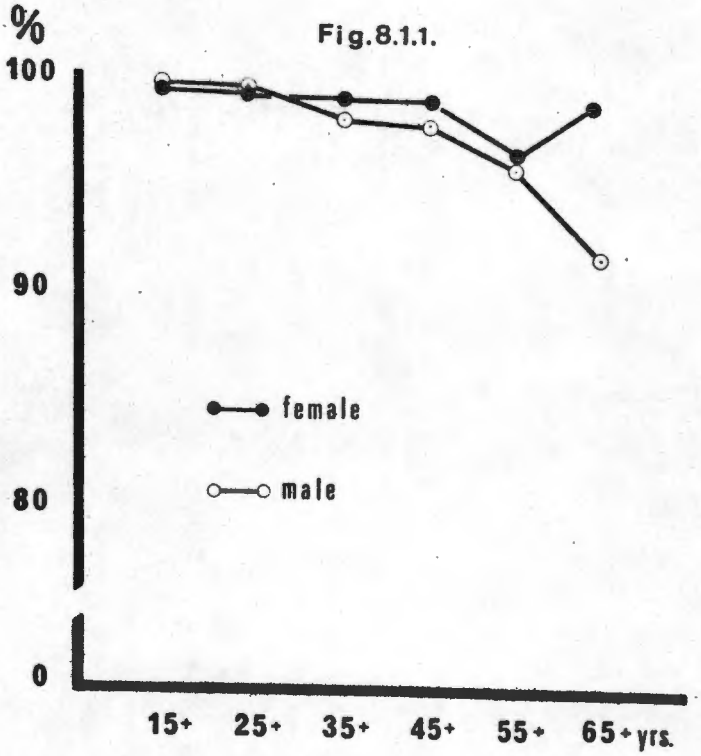


Fig.8.1.1.

DECREASE OF EXTERNAL ROTATION WITH AGE

External rotation in both shoulders decreased progressively in males so that by the 65+ decade almost 20% of the movement was lost. In females a different pattern was seen and there was only one decade when there was a marked reduction in external rotation. There was little difference in the two shoulders. The reason for the progressive decline in external rotation in the males are not clear, but it is similar to other reported findings where as much as 30% loss has been recorded in the 7th and 8th decades (De Palma 1973).

The senescent changes which occur in the shoulder joint have been fully described and include an increasing number of glenoid labral detachments, degenerative changes in the biceps tendon and tears in the fibrotendinous cuff. By the 7th decade 80% of all shoulders will show labial detachment and tears of the fibrotendinous cuff, with or without calcific deposits (De Palma 1973; Codman 1934). These calcific deposits are found in 8.0% of asymptomatic persons over the age of 30 years (Boyle 1969), and they must be regarded as part of the spectrum of degenerative changes in the shoulder. The decrease of external rotation with age is attributed to increasing fibrous tissue in the capsule of the shoulder joint which can be due to disuse (external rotation is not a commonly used movement) with resulting loss of elasticity and it is one of the reasons advanced for the lower incidence of shoulder dislocations in persons over the age of 45 years (De Palma 1973; Sokoloff 1980). These natural developments in the shoulder with increasing age raise the tautological arguments of what is a disease?

Shoulder disease in the Rietpoort population

For the purposes of the following discussion I have considered shoulder pain and impairment of function as a disease. Using this definition the prevalence of shoulder disease in the population is shown in Table 8.1.2.

TABLE 8.1.2.

THE PREVALENCE OF SHOULDER DISEASE

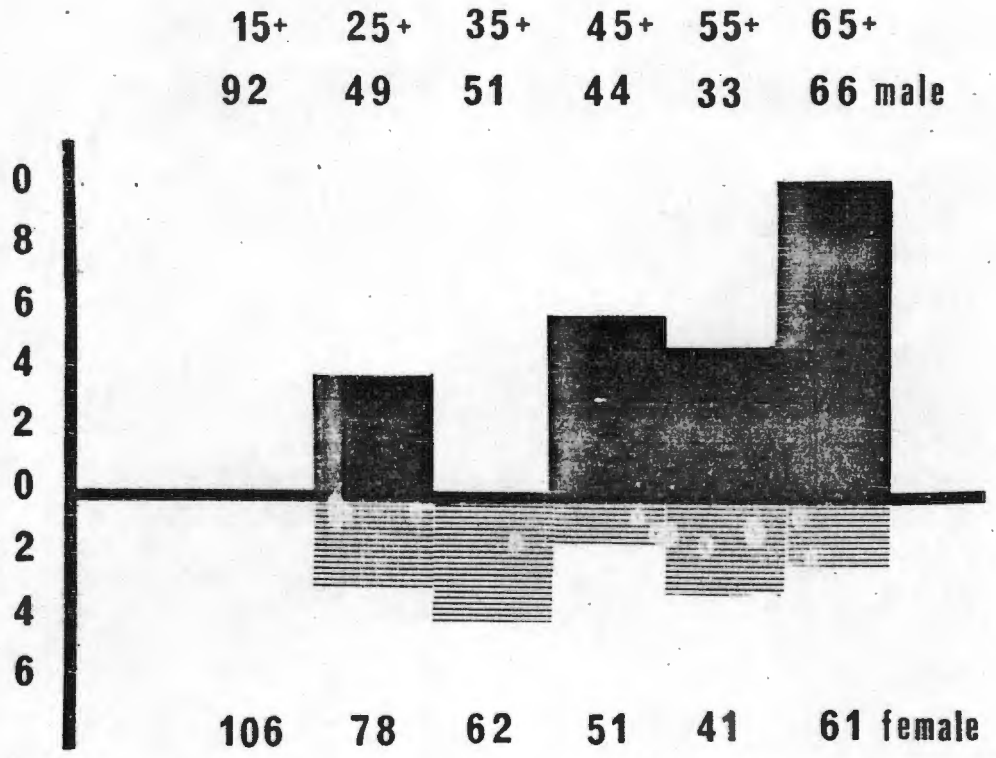
	15+	25+	35+	45+	55+	65+
MALES	0.0%	3.3%	2.0%	13.3%	15.2%	24.2%
FEMALES	0.0%	2.8%	4.8%	7.8%	9.8%	19.6%

The overall prevalence of shoulder disease is 7.0% of the population over the age of 15 years. The prevalence of shoulder disease is nearly equal in males and females up to the age of 45 years whereafter it rises steeply in males and by the 65+ decade 24.2% of males have shoulder disease. In females there is a slower progressive increase in the prevalence of shoulder disease up to the age of 65 whereafter it doubles in frequency. In the group 45+ which seem most at risk the prevalence was more than double that of the whole population while in the subjects over 65 years 1:5 persons (22%) had evidence of shoulder disease.

THE NATURE OF THE SHOULDER DISEASE

1.1. Lesions of the musculo-tendinous cuff

Disorders of the musculo-tendinous cuff (rotator cuff) especially lesions of the supraspinatus tendon were prominent and they were seen most frequently: (Figure 8.1.2.)



AGE-SPECIFIC PREVALENCE - SHOULDER TENDONITIS

Fig.8.1.2.

There were 36 persons who had a supraspinatus tendonitis which represents a prevalence of 4.9%. Males were affected twice as frequently as females (6.8% vs 3.2%). The prevalence increased with age so that by the age of 65 years 15.1% of males and 3.2% of females had a supraspinatus tendonitis. The right side was involved in 23 individuals, the left in 13 and both sides in 4. In one female aged 49 a bicipital tendonitis was found and in a male aged 76 there was a rupture of the long head of the biceps in association with bilateral supraspinatus tendonitis. In one female of 44 years and a male of 33 years the tendon lesion

could not be characterised and in both it was recorded as a rotator cuff lesion (in the female it was on the right side, and in the male it was bilateral). One other female (70 years) had a painful arc of movement which could not be characterised further. This may have had several causes including a tendonitis. Another female (66 years) had pain on abduction at 150^{0} suggesting an impingement of the supraspinatus tendon under the coraco-acromial ligament. The pathogenesis of supraspinatus tendonitis has been discussed by many eminent authorities (Codman 1934; Cyriax 1969; bland, Merritt and Boushey 1977; De Palma 1973; Mosely 1945). The first pathological event is believed to be thinning, fraying and fibrillation in Codmans area of critical hypovascularity. It is presumed that constant stress and the impingement of the rotator cuff against the coraco-acromial arch act in concert to produce a traumatic inflammatory reaction. In those tendons where calcium hydroxyapatite is deposited, a further source for acute inflammatory episodes is provided which does not require to be precipitated by trauma (Thompson, Ming Ting, Riggs et al 1968: McCarty and Gatter 1966; Swannell and Dixon 1966). Autoimmune reactions to denatured collagen and other structural proteins have also been suggested as a cause for recurrent inflammatory episodes. Some support for this hypothesis comes from studies on collagen in osteoarthritic joints where immunoglobulin deposits have been described in collagen tissues (Cook, Bennett and Ohno 1980; Cooke 1981). The studies of the antigenicity of disc material suggest that disc material is sequestrated from the immune system and evokes no reaction, but when it is exposed to the circulation it provokes an immune response (Elves, Bucknill

and Sullivan 1975; Gertzbein 1981). At least some of the tendon immunopathology may be due to cell mediated immune reactions (McNab 1973). To draw an analogy between the sequestrated disc and the collagenous tissue of Codmans area of critical hypovascularity is perhaps not far fetched (Rathburn and MacNab 1970). The increasing number of supraspinatus tendon lesions with increasing age would also fit in with ideas about ageing as an autoimmune disease (Walford 1962; Burnet 1970). However a primary mechanical or traumatic induced inflammation seems inescapable in view of the increased prevalence in males of supraspinatus tendonitis, but the two putative pathogenetic events are not mutually exclusive.

1.2. Adhesive capsulitis

There were five subjects who were considered to have this syndrome. All were in the 65+ age group. Three were males and two females. The basis for making this clinical diagnosis was that in all of these subjects there was a complaint of shoulder pain associated with a general reduction in all movements particularly marked in external rotation, abduction and less in internal rotation. In two of these subjects there was an associated fixed flexion deformity of the fingers of both hands suggesting the hand-shoulder syndrome. In both these subjects there was mild osteoarthrosis of the PIP joints but this would not have provided a suitable explanation for the flexed fingers. The right side was affected in 4 subjects and it was bilateral in one. There were no indications of the cause in these subjects (Hazleman 1972). One other female (69 years) with

diabetes mellitus had shoulder pain and painful restriction of external rotation which was regarded as either an incipient or recovering adhesive capsulitis. Attention has recently been drawn to the increased association of adhesive capsulitis in diabetes mellitus (Bridgeman 1972; Lequesne, Dang, Benasson et al 1977). Adhesive capsulitis is unique to the shoulder joint and it is currently believed to be a reflex sympathetic dystrophy. It is probable that it forms one end of a spectrum which includes the hand shoulder syndrome as the extreme form. There have been many theories of causation including bicipital tendinitis, or other shoulder tendonitis. and it seems that any condition causing pain and or immobility of the shoulder may be the initiating event (Thompson 1961). The pathology has been well described and is characterised by marked thickening and contracture of the capsule (Nevaiser 1945) due to new collagen deposition (Lindberg 1970). More recently an inflammatory reaction in the synovium and peri-articular tissues has been demonstrated in the hand - shoulder syndrome (Kozin, McCarthy, Sims et al 1976). Support for a localised immune reaction as a cause for the frozen shoulder comes from the demonstration that serum IgA and lymphocyte transformation is significantly depressed in patients with the condition (Bulgen, Hazleman, Ward et al 1978). In this Namaqualand population the serum immunoglobulins were measured in all the subjects with the frozen shoulder and in 20, age and sex matched subjects without overt shoulder disease, and in six age matched subjects with a shoulder tendonitis. The results are shown in the following table:

TABLE 8.2.1.

Levels of immunoglobulins in subjects with frozen shoulder, shoulder tendonitis and age/sex matched controls.

	Frozen shoulder (6)	Shoulder tendonitis (6)	Controls
IgG	2211.3mg %	2208.3	1923
IgA	510.0 (p= 0.008)	305.3	328.8
IgM	144.3	105.8	105.8

It can be seen that contrary to the other report, that IgA levels increased significantly (p= 0.008). No significant differences were found for IgG or IgM. When the group of age sex matched subjects with shoulder tendonitis were compared with the frozen shoulder subjects similar significant differences (p= 0.001) were found in the IgA levels. This is of some interest because it supports the idea of the role of immune reactions in the genesis of the frozen shoulder. The normal immunoglobulin levels in the tendonitis group suggests that a putative pathway via shoulder tendonitis (which is widely held) should perhaps be reassessed. Alternatively shoulder tendonitis may be an initiating event which is amplified by an immmune process. Further studies are needed to clarify this.

1.3. Unexplained shoulder problems

24 individuals (9 female, 14 male) were identified by an isolated reduction of external rotation. The mean age for this group was 62.95 years (range 33 - 81 years). The nine females had a mean

age of 60.4 years (range 33 - 70 years) and the males had a mean age of 65.7 years (range 43 - 81 years). The average range of motion was 0 - 57° for the right shoulder and 0 - 65° for the left shoulder. The reduction in external rotation was bilateral in 13, right sided in 6 and left sided in 4. In 16 subjects there was a history of shoulder pain and in 5 there was pain on passive external rotation. These subjects form a striking group because of the isolated impairment of external rotation which falls well below the range of external rotation for this population and the age group. The reason for this is not clear. In the fifteen subjects over the age of 65 years it may represent an extreme of the normal reduction of external rotation, but this is not likely in the younger subjects. A history of previous pain in the shoulder suggests that the reduction of external rotation may have been the result of some shoulder disease, possibly previous adhesive capsulitis, and this is supported by the fact that 6 of the subjects had a concomitant shoulder tendonitis. Another possibility is that they represent other previous shoulder disease e.g. old anterior capsular scars, such as may occur following anterior dislocation, (there was no history of this in these patients) or that it is due to disuse and/or the increased stiffness of collagen tissues with age (Verzar 1957; Ridge and Wright 1966). The two remaining causes for an isolated external rotation impairment are lesions of the infraspinatus tendon or a sub-coracoid bursitis (Cyriax 1969). It is also possible that some of these individuals may have had osteo-

arthrosis of the shoulder. One of these subjects was classed as having a generalised osteoarthrosis on the basis of involvement of 5 groups of joints (radiologically). Three individuals had no radiological osteoarthrosis, 5 had osteoarthrosis in 1 group of joints, 2 in 2 groups, 5 in 3 groups and 7 in 4 groups. In most the osteoarthrosis was mild (grade 2). Osteoarthrosis of the shoulder tends to be symptomless and moreover generally produces limitation of abduction rather than of external rotation (Cyriax 1969).

1.4. Previous shoulder trauma

One male and one female recalled previous trauma to the shoulder and one patient suffered recurrent dislocations of the right shoulder.

Shoulder involvement is clearly a common rheumatic manifestation in the population under study, and tendon lesions as a group were the most common cause of shoulder disease. It is of some interest to compare the prevalence of 4.9% in this study with data derived from general practice and specialist rheumatology practice. In an illness profile study among 15 general practitioners in Cape Town conducted over one year the incidence of tendonitis was 5.2% which includes tennis elbow (Silbert 1970) while in a specialist rheumatology practice the incidence of tendonitis/bursitis was 4.5% (Bohan 1981).

The increased prevalence of shoulder disease in the elderly does also hold less ons for those involved with the care of the elderly because shoulder pain and disease add a further dimension to the problems of the elderly and it may increase dependance. Furthermore when one considers that 3.9% of the population over 65 years had adhesive

capsulitis (frozen shoulder) in which prior shoulder pain and/or immobility of the shoulder are considered as important predisposing factors there is a great need to encourage early diagnosis and vigorous treatment.

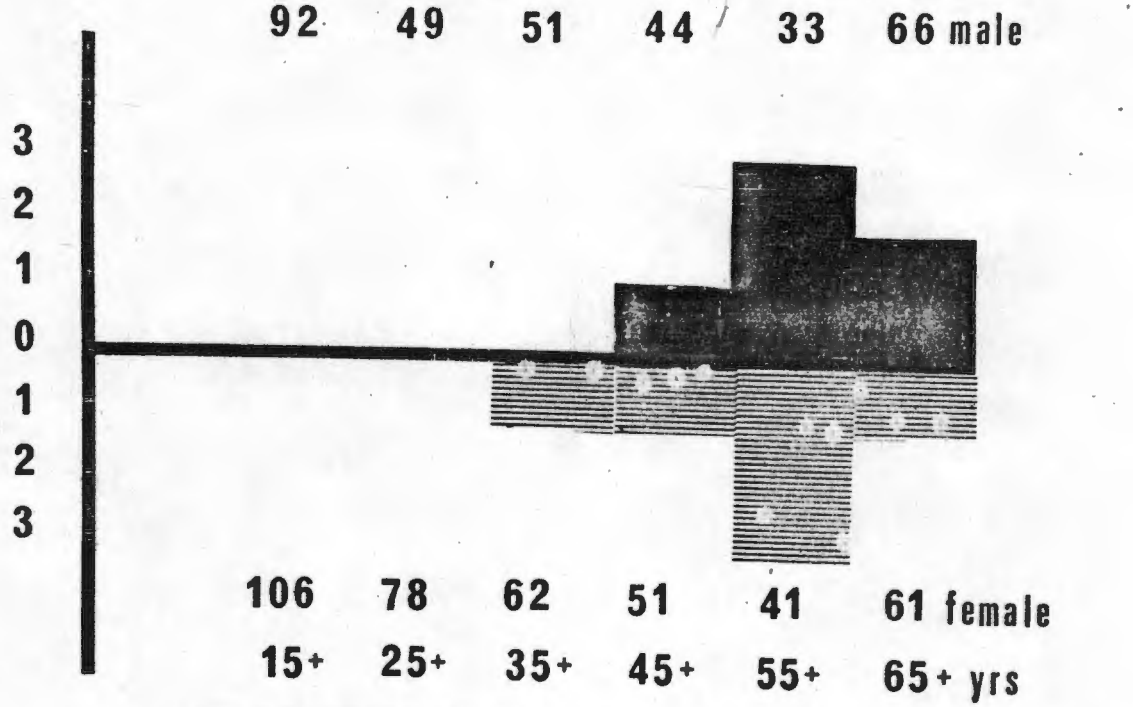
The factors which were important in the shoulder disease in this community were not clarified by this study but occupational factors were considered to be important. Other comparative studies will be needed to decide whether the population is particularly at risk. Preliminary data from an urban Coloured community suggests that the prevalence is lower and lends support to an occupational factor (Meyers 1980). It is also clear that a study of the epidemiology of shoulder disease is possible in a formal field study and that it presents no difficulty.

2.1. LATERAL EPICONDYLITIS (TENNIS ELBOW)

In this study lateral epicondylitis was diagnosed by finding local tenderness over the lateral epicondyle of the humerus and pain at this site which is provoked by resisted extension at the wrist while the elbow is held extended.

Twelve subjects fulfilled these criteria. There were 6 males and 6 females. The age specific prevalence is given in the following figure: (Figure 8.2.1.)

Fig. 8.2.1.



AGE-SPECIFIC PREVALENCE - LATERAL EPICONDYLITIS

The prevalence for adults over the age of 15 years was 1.86%. The age specific prevalence rose from 0.88% in the 35+ group to 8.1% in the 55+ age group. The right side alone was involved in 3 subjects, the left in 2, and it was bilateral in 7. In 5 of these subjects there was an associated shoulder disease (frozen shoulder in 1 and a supraspinatus tendonitis in 4). No association was found with De Quervains tenosynovitis or with the carpal tunnel syndrome. In 4 subjects there was an associated abnormality of the elbow i.e. a flexion deformity was found on the affected side in two individuals with no previous history of elbow disease/injury. In another subject with bilateral involvement

there was an associated reduction in the range of motion of the elbows, and in the fourth subject who also had bilateral epicondylitis, there was a history of previous elbow arthritis and both elbows showed a reduction in their range of motion.

There is a fairly comprehensive literature about lateral epicondylitis, but there is no record of an attempt to estimate the prevalence in a community. Most reports indicate that equal numbers of males and females are affected with a peak age incidence of 40 - 50 years (Garden 1961) and a tendency for the right side to be involved more frequently. The data from this study in general supports the clinical description but the peak age incidence is shifted to the 55 - 65 age group.

The causes of lateral epicondylitis

The first clinical description of epicondylitis is attributed to Runge (1873). The cause has not been elucidated and many aetiological factors have been suggested. Most authorities agree that repetitive activity especially pronation/supination with forced extension plays a role, but the mechanism of the pain which is produced has provoked a number of theories and an equally large number of different surgical procedures for curing the recalcitrant case. Up to 1936 at least 29 different pathological causes had been proposed (Cyriax 1936). The factors which have been suggested are trauma to the annular ligament of the radius leading to an inflammatory reaction (Bosworth 1955; Bosworth 1965; Boyd and McLeod 1973), periostitis of the conjoined tendon, (Goldie 1964) tears in the conjoined tendon (Cyriax 1936; Romer 1922), entrapment neuropathy

of the radial nerve (Roles and Maudsley 1972). This very wide variety of putative pathogenetic mechanisms prompted Kellog-Speed to write 'the aetiology of tennis elbow is various, its pathology obscure and its cure uncertain' (Kellog-Speed 1929). Fifty years later we are no further, but this is perhaps because there are so few resistant cases that the common cause has eluded investigators. This is probably a heterogenous condition which the varied numbers of successful surgical procedures attest to. Three types of epicondylitis are described, the teno-periosteal type which is the most common, the intermuscular variety and the subcondylar type (Cyriax 1969). In one study of lateral epicondylitis, attention was drawn to the fact that 38% of cases have other soft tissue lesions, notably tendon lesions at the shoulder, De Quervains tenosynovitis and trochanteric bursitis (Boyd and McCloed 1973). A shoulder lesion was present in 5 of the 12 subjects in the survey, but the meaning of this is not clear since there was a high prevalence of shoulder lesions in the age group where the peak prevalence was found for epicondylitis. Only one subject had a medial epicondylitis.

3.0. OTHER SOFT TISSUE LESIONS

3.1. Bursitis

Olecranon 'bursitis' was diagnosed by the finding of swelling of the olecranon bursa at the tip of the olecranon. Two males aged 73 and 43 had an olecranon bursitis which gives a prevalence of 0.27%

of the adult population. In the one subject it was bilateral and in the other it was on the right side. The cause of the bursitis was not established in either subject since none of the common causes such as rheumatoid arthritis or gout were present. The bursal enlargement was asymptomatic in these subjects. No examples of other forms of 'bursitis' were encountered. The biology of the subcutaneous and deep bursae has recently been reviewed (Canoso 1981).

3.2. Dupuytren's contracture

Dupuytren's contracture was noted in 7 individuals (5 males and 1 female). The mean age of these subjects was 69.1 years (47 - 83 years). This represents a prevalence of 0.95% of adults. All the male subjects were or had been heavy manual labourers. There was no evidence of the common causally related diseases such as chronic liver disease, alcoholism or diabetes mellitus in any of these subjects (Spring, Fleck and Cohen 1970). The prevalence in Caucasians is given as 1 - 3% (Viljano 1973). Although it is tempting to suspect heavy manual labour as a cause this has not been borne out by other studies (Hueston 1963; Wielinga 1961). Moreover it should have been seen more frequently in this if an occupational factor such as heavy manuel labour was important.

3.3. Flexor tendon disease of the fingers

One male subject age 77 had crepitus and tenderness over the right middle flexor tendon. There was no associated disease, and no history of trauma. A second subject a female aged 60 years had a

flexor tendon nodule in the thumb flexors for which no cause was found, and a male of 59 had a swanneck deformity of the right index finger due to an old severed superficialis tendon.

3.4. De Quervains tenosynovitis

De Quervains synovitis was diagnosed if the subjects complained of pain at the lower end of the radius in the anatomical snuff-box associated with local tenderness and a positive Finkelstein test. Only 2 subjects were found which represents a prevalence of 0.27%. Both these subjects were females aged 59 and 68 years respectively. The older woman had an associated soft tissue problem of the shoulder, but the relevance of this finding is in doubt.

3.5. Carpal tunnel syndrome

A clinical diagnosis of carpal tunnel syndrome was made when there was a history of pain and/or numbness and paraesthesia in the hand at night and which was relieved by exercise, and a positive Phalen test (Phalen 1966). Two females age 54 years and 63 years were considered to have a carpal tunnel syndrome. There was no associated disease such as wrist arthritis, rheumatoid arthritis, hypothyroidism. The diagnosis of carpal tunnel syndrome rests on the characteristic symptoms and it can be confirmed electrophysiologically. Studies using only motor conduction across the carpal tunnel give a low yield of positives but with more refined sensory conduction this can be useful to aid in the diagnosis (Kemble 1968). The problem is not always easy to diagnose and it may be confused with the

sublimus syndrome (Gardner 1970) and in some the syndrome may need encroachment on the nerve at the intervertebral foramen for its full expression, the so called double crush phenomenon (Upton and McComas 1973)

3.6. Fibromyalgia/Fibrositis

Two individuals were seen in whom the fibromyalgic syndrome was diagnosed on the basis of localised muscle tenderness and irritability associated with muscle spasm. This is probably an under estimate of the frequency of the condition.

3.7. Polymyalgia rheumatica

No examples of polymyalgia rheumatica were found.

The data from this study shows that 65 of the 734 adult subjects had a non-articular soft tissue lesion. This represents a prevalence of 8.85%.

Rheumatic disease recorded in the Mission hospital records

An examination of the Mission hospital records shows that during 1978, 72 patients were recorded as having a rheumatic problem, this is 2.07% of all the diagnoses. It is likely that most of the hospital visits for rheumatic problems were for osteoarthrosis, because the soft tissue rheumatic problems tend to be self-limiting. Their potential for producing disability, albeit for a limited period, is however unquestioned. Their importance in an elderly population has already

been discussed. The men and women of this population have been labourers and domestics for most of their lives. Life in this arid environment is hard and survival makes more demands on the musculoskeletal system up to extreme old age. There are no comparable studies of soft tissue rheumatic disease in other individuals living in cities where amenities provide the musculoskeletal system with less stress. A major deficiency in an evaluation of soft tissue rheumatic problems is the lack of easily applied criteria for use in field studies and if it were for instance possible to estimate the prevalence of the fibromyalgic syndrome accurately it is likely that the numbers of rheumatic problems would be increased well beyond that which has been produced in this study.

CHAPTER 9

OSTEOPOROSIS

OSTEOPOROSIS

OSTEOPOROSIS

Semantic problems

Two terms are used to describe a reduction of bone mass: osteopaenia which is used to describe the loss of bone mass, and osteoporosis which describes the structural failure of bone consequent upon a reduced bone mass (Hahn and Hahn 1976). Osteopenia is still not universally accepted and osteoporosis is still widely used to describe both conditions. Several definitions have been advanced for osteoporosis which range from "too little bone of normal composition which results from a failure of osteoblasts to lay down bone matrix" (Albright 1947) to "insufficient structural, non reactive, non exchangeable bone mass" (Urist 1964). Osteoporosis is characterised by a loss of trabecular bone and thinning of cortical bone in both the axial and the appendicular skeleton.

The physiology of bone

Bone serves a dual function in man. It provides a means of support and locomotion, while at the same time serving as a reservoir of ions which are important in several metabolic processes. This latter function takes precedence over the supportive function. The major regulating factors of bone metabolism are physical/mechanical stress: the concentration of calcium and phosphorus in the extra-cellular fluid, various hormones such as parathormone, calcitonin and several biologically active metabolites of Vitamin D. Bone is composed of a matrix of collagen cross-linked in pentagonal

bundles of microfibrils and a mucopolysacharide ground substance, plus mineral in the form of hydroxyapatite crystals. The apatite crystals nucleate at specific sites on the collagen matrix, between adjacent fibres. This arrangement is in two morphological forms, the spongiosa, and cortical or compact bone. The most metabolically active part of the bone is the spongiosa and it is the first portion of bone to respond to hormonal stimuli. Bone turnover rate varies according to age and it represents the equilibrium between formation and resorption.

Formation is the function of osteoblasts which lay down the collagen matrix along lines of mechanical stress induced by piezo-electric forces (Harris and Heaney 1969). Mechanical stress is the major stimulus to osteoblastic activity. Resorption of bone is the function of osteoclasts which are influenced by several hormones.

1. Parathormone (PTH)

The effects of PTH are:

(a) To activate osteoclasts leading to bone resorption with the liberation of calcium;

(b) To decrease renal tibular excretion, increase urinary phosphate;

(c) To increase intestinal calcium absorption;

2. Calcitonin (CT)

(a) Decreases bone resorption.

(b) Increases calcium excretion in the urine.

(c) Decreases intestinal calcium absorption.

The effect of CT is dependant on the turnover rate of bone i.e. it is most marked where bone turnover is high.

3. Vitamin D and its metaboliles

The function if Vit.D. is to increase the availability of calcium and phosphate for bone formation.

Osteoporosis is a radiological diagnosis of reduced bone mass.

Histologically three types of bone loss are:

(1) A parallel loss of bone mineral/matrix.

(2) Osteomalacia in which deficient bone mineralisation is the dominant finding.

(3) Osteitis fibrosa cystica where osteoclastic resorption of bone occurs with replacement of normal bone by fibrous tissue.

The following classification reflects these 3 histological types, but the problems are compounded by the frequent occurrence of mixtures e.g. in the elderly osteoporosis/osteomalacia may co-exist.

Classification of generalised osteoporosis

1. Osteoporosis (Parallel loss of mineral and matrix).
   1. Ageing
   2. Immobilisation
   3. Premature menopause

2. Osteomalacia (inadequate mineralisation).
   1. Vit.D. deficiency
   2. Phosphate wasting syndromes

3. Osteitis Fibrosa cystica (PTH induced mineral/matrix resorption).
   1. Primary hyperparathyroidism
   2. Secondary hyperparathyroidism

4. Corticosteroid induced osteoporosis

5. Other disorders - thyroid disease, malignancy.

The importance of osteoporosis

The importance of osteoporosis lies in the structural failure of bone which is presumed to be a consequence of decreased bone mass. If a definite relationship between bone mass and the occurrence of fractures exists it would be axiomatic that accurate measures of bone mass should be developed and if it can be further shown that a critical level of bone mass exists for a given individual in relation to height and weight, it would also be of the greatest importance to determine the threshold values below which the risk of fractures is sufficiently high to warrant treatment (Cohen, Aloia and Letteri 1978).

Methods for evaluating osteoporosis

1. Radiogrammetry (Radiomorphometry)

A number of radiographic measurements have been used to measure osteo-

porosis. All suffer from the major deficiency that considerable bone mineral loss (30 - 60%) will have occurred before the radiographs will show this loss (Lachman 1955). The radiographic methods which have been used are:

1.1. General

A simple 0 - 4 grading of osteoporosis of hands and feet has been used in comparison with a set of standard graded films (Kellgren and Bier 1956). This method has yielded fairly consistent results with little inter-observer error and with only modest discrepancies. Osteoporosis measurements of the spine are much more difficult although a grading system has been developed (Donaldson and Nassim 1954; Kamiyama, Kobayashi and Takahashi 1968; Nordin 1960).

1.2. Cortical thickness measurements

A decrease in the thickness of cortical bone is a constant feature of senile osteoporosis and measurements of metacarpal cortical thickness has been used to determine the presence of osteoporosis (Barnett and Nordin 1960; Nordin, MacGregor and Smith 1966). Subsequent studies have shown that the index of cortical area gives a better correlation with ash/unit length than other indices of bone quantity (Exton-Smith, Millard, Payne et al 1969; Dequeker 1976) and others have suggested that relating cortical thickness to total area is a better method (Evans, McDonnel and Schieb 1978). Similar methods and refinements have been used with different bones such as

the lower end of the radius (Meema and Meema 1969) and the hip (Singh, Riggs, Beabout et al 1973). In addition to the methodological problems of radiogrammetry, there is another difficulty that the measurements of osteoporosis in the appendicular skeleton do not necessarily reflect similar changes in other bones. Thus although an association between the cortical thickness of the shaft of the radius and spinal porosity has been demonstrated (Saville 1967) there are some forms of osteoporosis where the long bones escape (Dent 1955).

1.3. Osteodensitometry

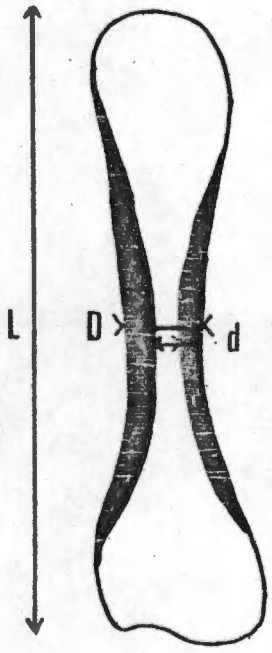
This is an isotopic method using a monoenergetic radiation source which provides a much more accurate reflection of bone mass (Cameron, Mazees and Sorenson 1968; Wahner, Riggs, Beabout 1977).

1.4. Neutron activation analysis provides another method for measuring bone minimal density, but the technique requires sophisticated apperatus (Cohen, Ellis, Wallach et al 1974).

The evaluation of osteoporosis in the Rietpoort population

In the Namaqualand population two measurements of cortical thickness were used, the area index and the ratio cortical area to surface area. The following diagram indicates the metacarpal measurements used:

Fig.9.1.1.



$$\text{AREA INDEX} = D^2 - d^2$$

$$\text{RATIO} = \frac{D^2 - d^2}{DL}$$

METACARPAL MEASUREMENTS

All measurements were made on the 2nd right metacarpal. The length was measured with a millimeter rule and the measurements D and d were taken at the mid point. These two distances were measured with a Vernier Caliper (accurate to 0.01 mm). The area index was used because it provides a good correlation with the ash weight of bone and the ratio was used because the introduction of surface area measurement introduces some standardisation and allows for better comparison between different individuals of the same and of the opposite sex (Exton-Smith, Millard, Payne et al 1969).

The age specific area index/metacarpal ratio

The age specific prevalence for the area index is given in the following figure: (Figure 9.1.2.)

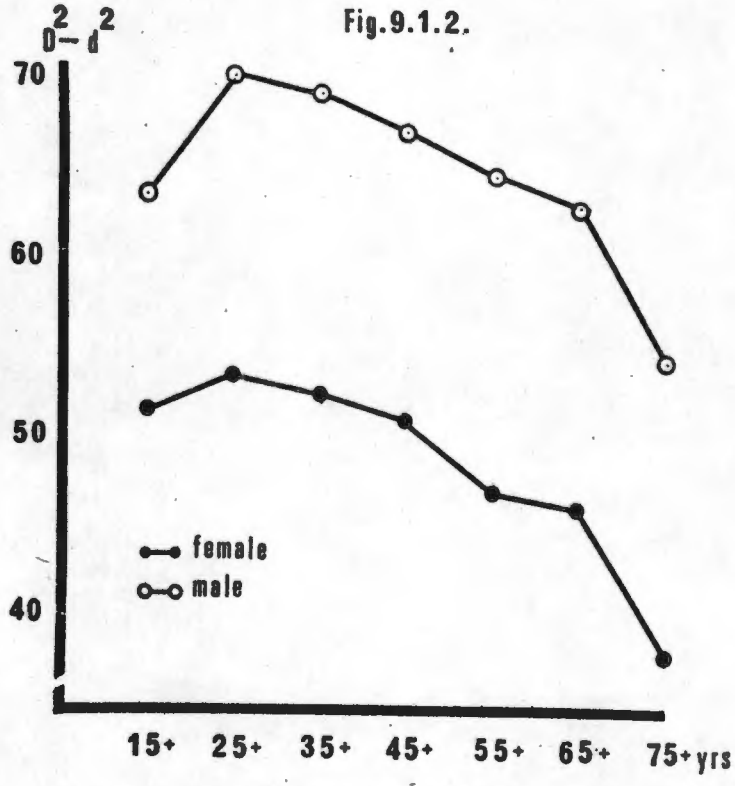


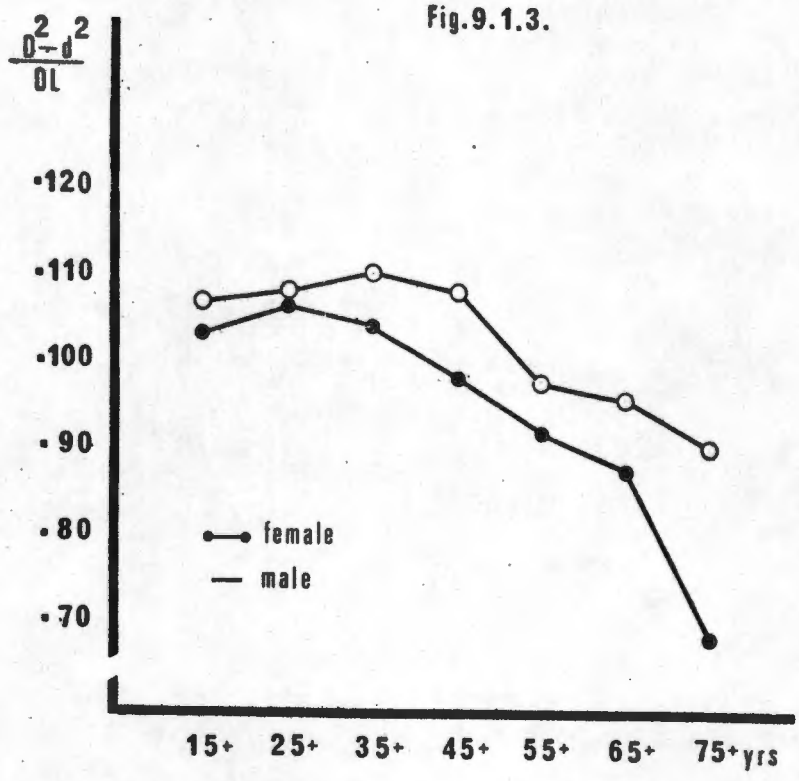
Fig.9.1.2.

AGE-SPECIFIC PREVALENCE - AREA INDEX

In males there was an increase in cortical area from the 15+ to the 35+ decade, whereafter it decreased progressively with increasing age. The changes with each decade were not statistically significant up to 65+ and 75+ decade when a significant reduction of cortical area was found ($p= 0.02$). In females the cortical areas were smaller than in men ($p= 0.001$). The measurements increased up to the 35+ age group and then started to decrease ($p= 0.04$) and further significant reductions were seen in those aged 75+ years ($p= 0.001$). This data can be compared with the

metacarpal ratio data in the following figure: (Figure 9.1.3.)

Fig.9.1.3.



AGE-SPECIFIC METACARPAL RATIO PER DECADE

The metacarpal ratios showed essentially the same trends and their use made it possible to compare the male and female data. There were no significant changes in the males, while in females there is still a significant decreased bone mass in the subjects 75 years and over.

The prevalence of osteoporosis in the Rietpoort population

The mean metacarpal ratio for the whole population was 0.102±14.7. Osteoporosis was defined as all values which fell 2 standard deviations below this mean. This defined 7 males and 15 females who

were osteoporotic and it represents a prevalence of 4.0% for males and 6.5% for females. The age specific prevalence of osteoporosis is shown in the following table:

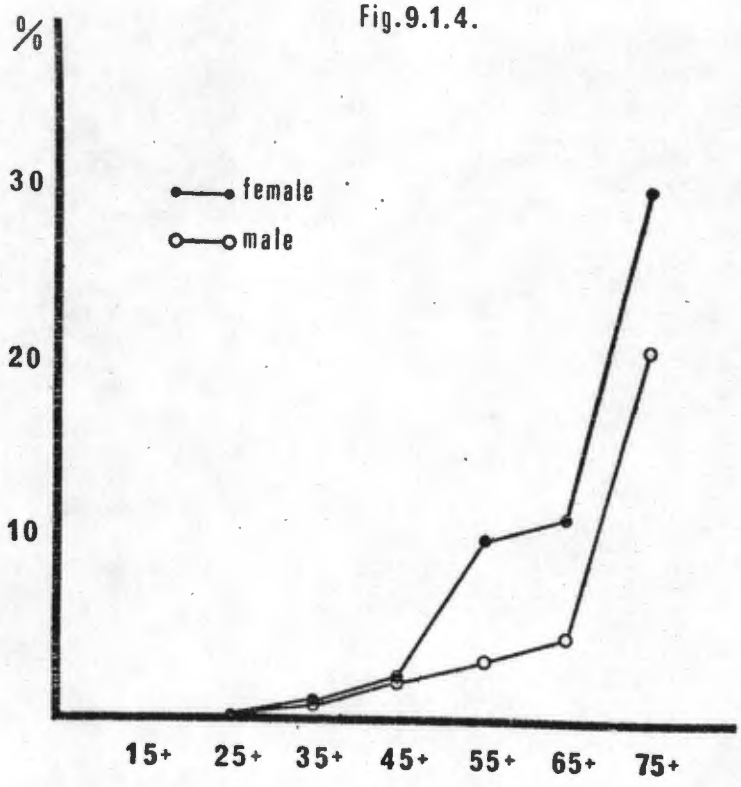
TABLE 9.1.

The age specific prevalence of osteoporosis in males/females

	MALES			FEMALES		
AGE	NO:	NO: POSITIVE	%	NO:	NO: POSITIVE	%
15+	36	0	0.0	51	0	0.0
25+	23	0	0.0	40	0	0.0
35+	27	0	0.0	38	0	0.0
45+	29	0	0.0	39	2	5.0
55+	16	1	6.3	24	3	12.5
65+	23	2	8.7	25	1	4.0
75+	20	4	20.0	15	9	60.0

From this data it is clear that the older age group made the contribution to osteoporosis in the population and the females over 75 years showed three times more osteoporosis than the males of a similar age. Another way in which the contribution of age can be looked at is to measure the percentage loss of bone for each decade. In order to do this the maximum bone mass attained for males and for females was taken as the reference point (i.e. the age group 25 - 34 years). The percentage decrease of bone was calculated for each decade. The percentage loss of bone/decade is shown in the following figure: (Figure 9.1.4.).

Fig.9.1.4.



PERCENT LOSS OF BONE MASS PER DECADE

The percentage loss of bone was nearly equal in males and females up to 55 years whereafter it rose in females so that by 75+ years 35% of the bone mass has been lost. In the males the percentage of bone loss was slower and by the 75+ decade the percentage loss was 21%. The progressive loss of bone mass with increasing age is regarded by most authorities as a universal one which starts from the age of 40 - 50 years.

Comparison with other studies and the relationship to fractures

The data from Namaqualand parallels, that of other published series (Solomon 1979, Garn, Rohmann, Wagner et al 1967). The factors which are contributory have been well discussed in several excellent

reviews (Avioli 1977; Meema and Meema 1969; Saville 1967). The greatest importance of osteoporosis must be in its putative relationship to fractures of the axial and appendicular skeleton. There is compelling evidence which suggests that there is an inverse relationship between bone mass and fractures (Iskrant and Smith 1969; Solomon 1968; Nordin 1971; Chalmers and Ho 1970). Some of the difficulty in accepting a direct causal role of osteoporosis is the observation that there is a non-linear relationship between the two conditions (De Quecker 1976, Bauer 1960) and the observations that the rate of cortical/trabecular loss is the same whether the patients are symptomatic (fracture of vertebral body) or asymptomatic (Smith, Johnston and Yu 1972). Studies from South Africa have shown a marked disparity between fractures of the femoral neck in Black and White South Africans but both groups show essentially similar degrees of bone loss with age (Solomon 1979). An examination of the hospital records of the Mission Hospital at Rietpoort does not show any record of femoral neck fractures, and none of the subjects reported previous femoral neck fractures. It is not likely that this could have been missed but a much more careful evaluation will be needed to relate the osteopenia in the population to fractures. What contribution the osteoporosis in this population made to back-ache (see chapter 5) is conjectural. This study has also not attempted to define the causes of the osteoporosis, but its occurrence in the aged suggests that most if not all was involutional, although osteomalacia may have been present in some.

CHAPTER 10

CAMPTODACTYLY, CLINODACTYLY, BONE AND JOINT TRAUMA HALLUX VALGUS AND ARTICULAR HYPERMOBILITY

CAMPTODACTYLY, CLINODACTYLY, BONE AND JOINT TRAUMA HALLUX VALGUS AND ARTICULAR HYPERMOBILITY

10.1. CLINODACTYLY

Clinodactyly is the result of shortening of the middle phalanx of the fifth finger in its radial aspect which results in radial deflection of the terminal phalanx. It is considered to be the result of incomplete ossification (Anderson and Klintworth 1961). In this study there were 12 adults (7 males and 5 females) and two children affected. This gives a prevalence of 1.09% for the whole population and 1.63% for adults over the age of 15 years. The prevalence in this group is higher than that of the study in Leigh, Wensleydale and Watford, where a prevalence of 0.3% was recorded (Lawrence 1977c). This study also differs from the others in the larger numbers of males affected. The deformity is inherited as an autosomal dominant and in this population an affected father passed the deformity on to two of his four children.

10.2. CAMPTODACTYLY

This condition of flexed fingers is confined to the fifth digit and is thought to be due to shortening of the superficalis tendon (Hegner 1924). It was found in 1.9% of surveys in Leight, Wensleydale and Watford where it occurred with equal frequency in males and females (Lawrence 19774). In the present study there were 10 females affected and 5 males, giving a prevalence in adults of 1.17%. There is a genetic influence and it may be inherited as an

autosomal dominant (Spaar 1964; Temtamy and McCusick 1978; Welch and Temtamy 1966). In this population study no familial associations were found.

10.3. BONE AND JOINT TRAUMA

Trauma to the musculoskeletal system can be expected in a population where occupations are heavily dependant upon manual labour. Most of the trauma in the Namaqualand study was found in the males. The following Table 10.3.1. provides an analysis of the bone and joint trauma in this population:

TABLE 10.3.1.

INJURY TO BONES/JOINTS IN ADULTS

1. BONE INJURY	NO:	%
SHOULDER	2	11.8
HAND (THUMB/FINGERS)	5	29.4
RADIUS	4	24.0
ELBOW	1	6.0
FOOT	1	6.0
LUMBAR SPINE	4	24.0%
	17	

2. JOINT INJURY	NO:	%
ELBOW	6	33.3%
HAND (ALL JOINTS)	8	44.4%
RADIOCARPAL	2	11.0%
KNEE	1	6.0%
ANKLE	1	6.0%
	18	

Injuries to bones were most frequent in the lumbar spine, (by history) the radius and the bones of the hand, while the joint injuries were most commonly encountered in the hands and elbow. The prevalence of recalled trauma was 4.76% of the adult population who were seen. In addition there were 27 subjects whose hand radiographs showed evidence of past trauma which was not recalled. When these subjects are added to those who recalled previous trauma the prevalence rose to 8.4%. (For discussion of the role of trauma in osteoarthrosis of the hand see chapter 7). The importance of chronic joint disease as a cause of disability in agricultural workers was first noted in Sweden (Kahlmeter 1932) when agricultural pensioners were compared with industrial and other pensioners. Rheumatic complaints are also much higher in agricultural workers than other professional groups in Britain (Brooke 1953; Lawrence 1977e) and in the United States (Woolsey 1952) but as a group British agricultural workers were less often off work (Lawrence 1977e). A much higher incidence of disc prolapse has been documented in agricultural workers. There are no studies on agricultural workers in South Africa with which to compare the Namaqualand data but such comparison would in any case prove difficult because differences in farming techniques could influence such comparisons. It does seem reasonable to suggest that trauma to the musculoskeletal system in this population is at least one important causative factor in some of the rheumatic problems encountered in the population.

10.4. HALLUX VALGUS

Hallux valgus is a common finding in elderly white females, and it

is less common in Black females (Gottschalk, Sallis, Beighton, et al 1980; Narmocpt amd Jardu 1955). This condition was evaluated in all subjects of the Namaqualand population in whom radiographs were available. Two hundred and seventysix (276) foot radiographs of males, and 308 foot radiographs were measured for hallux angles, intermetatarsal angles and forefoot angles. The mean age specific hallux valgus angle is shown in the following Table 10.4.1.

TABLE 10.4.1.

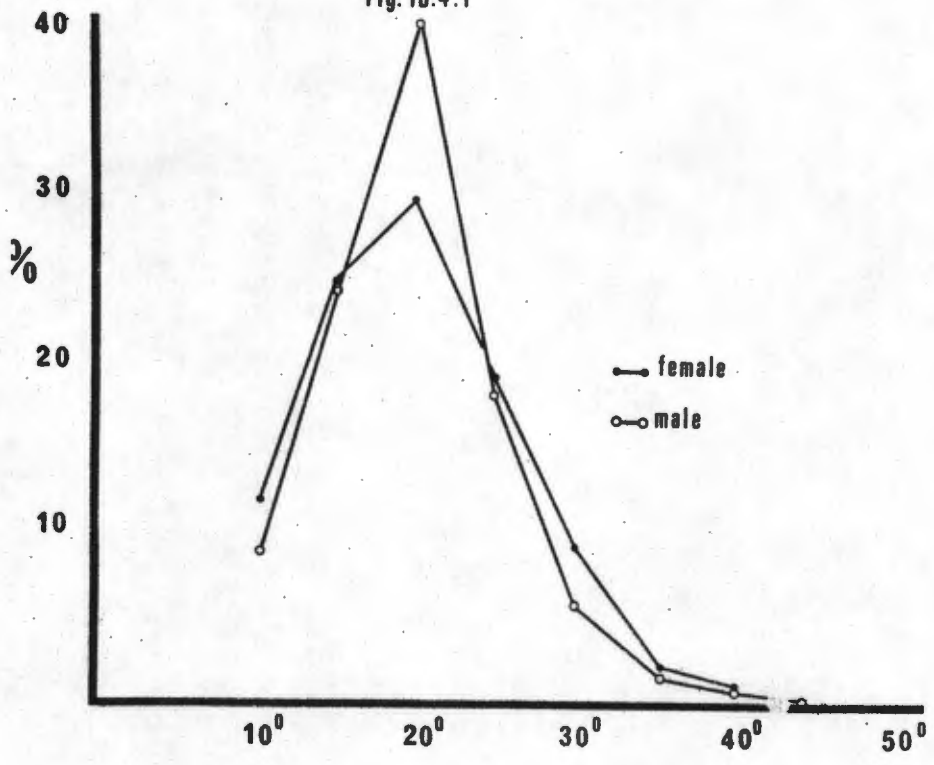
MEAN AGE-SPECIFIC HALLUX ANGLES IN MALES AND FEMALES

AGE	FEMALE			MALE		
	NO:	R	L	NO:	R	L
15+	64	$15.78^{0}\pm0.68$	$16.96^{0}\pm0.63$	51	$18.55^{0}\pm0.73$	$18.66^{0}\pm0.73$
25+	66	$19.21^{0}\pm0.68$	$18.71^{0}\pm0.64$	44	$16.85^{0}\pm0.83$	$16.61^{0}\pm0.70$
35+	57	$18.25^{0}\pm0.94$	$17.26^{0}\pm0.79$	52	$17.28^{0}\pm0.83$	$16.63^{0}\pm1.0$
45+	46	$17.83^{0}\pm1.06$	$18.05^{0}\pm0.07$	41	$17.91^{0}\pm0.83$	$18.10^{0}\pm1.13$
55+	32	$16.11^{0}\pm1.42$	$17.23^{0}\pm1.56$	28	$17.16^{0}\pm1.31$	$17.33^{0}\pm1.5$
65+	29	$17.17^{0}\pm1.56$	$18.36^{0}\pm1.56$	32	$16.93^{0}\pm1.16$	$17.73^{0}\pm1.28$
75+	14	$18.89^{0}\pm2.21$	$18.67^{0}\pm1.92$	28	$19.26^{0}\pm1.94$	$17.51^{0}\pm1.28$

There was a significant difference between the hallux angles of the right foot in females when the 15+ age group was compared with the 25+ age group ($p= 0.001$) but in succeeding decades no significant differences were found. In the males there was a significant difference between

th 15+ and 25+ age group. Hallux angle was larger in males than in the females in the 15+ age group (p= 0.008). The frequency distribution of hallux angles (right foot) shows that 73% of the males and 68% of the females have hallux angles of less than 20^0 (Figure 10.4.1.).

Fig. 10.4.1

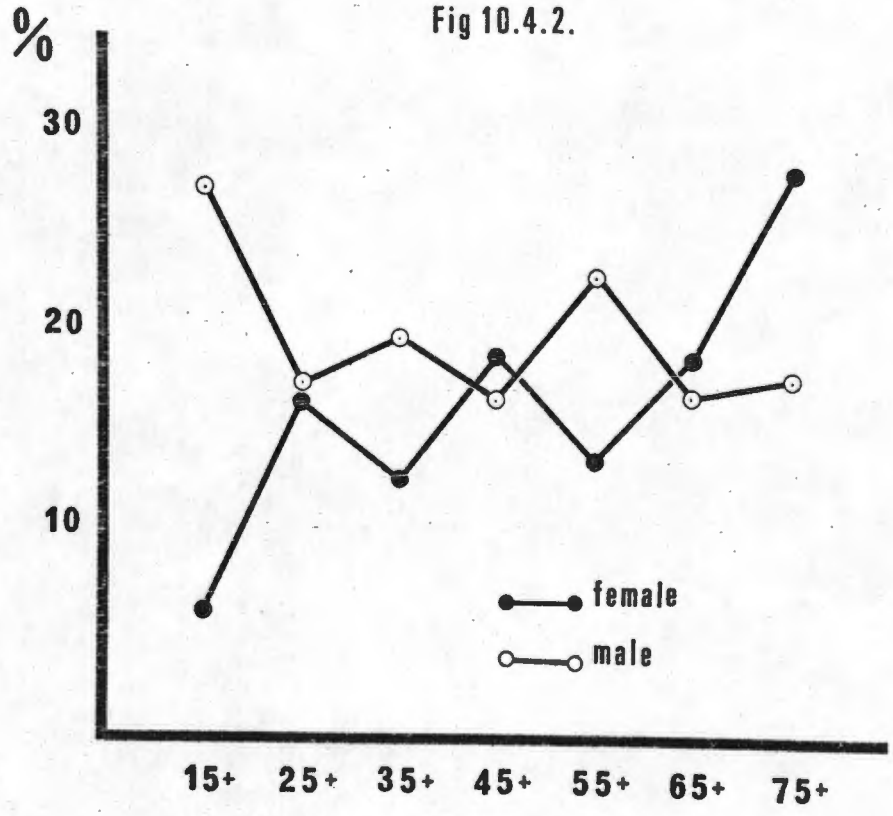


FREQUENCY DISTRIBUTION OF HALLUX VALGUS ANGLE

The mean hallux angle for the right and left foot was 17.56±1.38 and 17.69±1.28 for females and 17.69±0.69 and 17.5±0.65 for the males respectively. Two standard deviations were chosen to define the limits of normality in this population. The normal range of hallux angles for females was thus 10.7^0 - 24.5^0 and 14.2^0 - 21.2^0 for males.

The application of this definition showed that excessive valgus angles were found on the right and left in 43 and 46 instances in females and in 56 and 59 instances in males. The overall prevalence of hallux valgus was 14% for females and 20.8% for males. The age specific prevalence of hallux valgus is shown in the following Figure 10.4.2.

Fig 10.4.2.

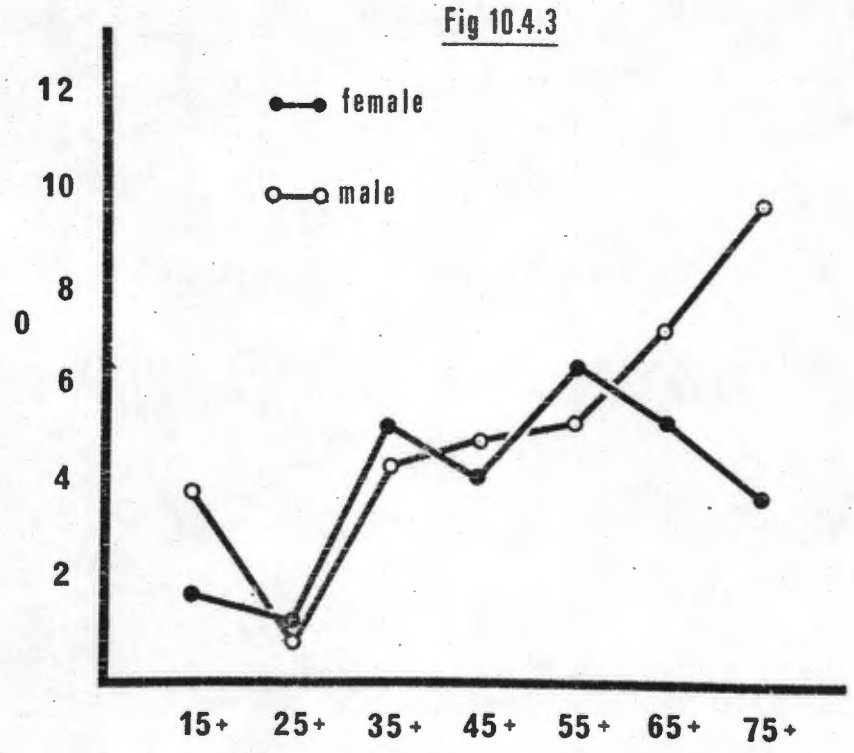


AGE-SPECIFIC PREVALENCE OF HALLUX VALGUS

The age specific prevalence for hallux valgus was maximal for young males and this decreased to levels of 15 - 20% in each succeeding decade. In females the hallux valgus prevalence started at 5% in the younger females and increased progressively to a maximum in the

subjects over 75 years. In the males and the females in the 15 - 24 year age group the abnormal valgus angles were $5^{o}$and 1.9^{o}. This deviation from the defined normal limits was less in the 25 - 34 year age group and thereafter the angles increased in males and females up to the 55 - 64 year age group. After this the hallux angles decreased in the females and continued to rise in the males. These changes are demonstrated in the following Figure 10.4.3.

Fig 10.4.3



CHANGES IN ABNORMAL VALGUS ANGLE WITH AGE

The hallux valgus angles in this population are larger than have been reported (Hardy and Clapham 1951). Hallux valgus was found more

frequently in males in Namaqualand which is surprising, but an examination of some of the published reports shows that an almost equal occurrence in both sexes is claimed (Hardy and Clapham 1951; Johnson 1956). The Namaqualand study does not relate these hallux valgus changes to symptoms, where it is clear that there is a difference between males and females.

The intermetarsal angle - metatarsus primus varus

The age specific prevalence of the intermetarsal angles is shown in the following Table 10.4.2.

TABLE 10.4.2.

MEAN AGE - SPECIFIC INTERMETARSAL ANGLES FOR MALES/FEMALES

AGE	NO:	FEMALE		NO:	MALE	
		R	L		R	L
15+	64	9.17±0.27	8.96±0.31	51	8.53±0.32	8.69±0.37
25+	66	9.06±0.33	9.01±0.26	44	8.28±0.38	8.67±0.39
35+	57	8.57±0.38	8.35±0.32	52	8.28±0.30	8.67±0.38
45+	46	8.84±0.48	8.58±0.44	41	7.73±0.30	8.70±0.30
55+	32	9.18±0.43	8.92±0.53	28	8.00±0.47	7.76±0.26
65+	29	8.84±0.58	9.39±0.66	32	8.03±0.52	7.56±0.50
75+	14	9.46±0.88	8.78±0.95	28	8.28±0.62	8.28±0.49

The mean intermetatarsal angle was 8.9±0.51 (2xSEM) and 887±0.65 (2xSEM) for the right and left foot respectively for females (upper limit of normal 9.9^{o} and 10.1^{o}), and 8.25±0.35 (2xSEM) and 8.27±0.37 (2xSEM).

upper limit of normal for right and left foot 8.93^{0} and 8.99^{0}) respectively for the right and left foot in males. Intermetarsal angles greater than the upper limit of normal were found on the right side in 116 and on the left in 98 instances in females. This represents a prevalence of 37.6% and 31.8% respectively. In males the prevalence was 26.4% and 30% for the right and left foot respectively. Considerable variation in the age specific prevalence of abnormal intermetarsal angles was found in both the males and the females (see appendix 10.4.2.). Abnormally large intermetarsal angles were most frequent in the 15+ decade. This may be partly due to the fact that up until early adulthood shoes are not worn frequently. The upper limit for the intermetarsal angles agree with other published reports (Gottschalk, Sallis, Beighton et al 1980; Mitchell, Fleming, Allen et al 1958; Carr and Boyd 1968). The correlation co-efficient for intermetarsal angles and abnormal hallux angles for females was r= 0.53 and for males it was r= 0.49. In females there was a significant association of hallux angles of 25^{0} - 29^{0} and 30^{0}+ with an increased intermetarsal angle (p= 0.001 and p= 0.05). When the population was divided arbitrarily into two groups i.e. 15-44 years and 45+ years the differences continue to remain significant.

The forefoot angle

The forefoot angles are shown in the following Table 10.4.3.

TABLE 10.4.3.

MEAN AGE SPECIFIC FORE-FOOT ANGLES IN MALES AND FEMALES

AGE	NO:	FEMALE		NO:	MALE	
		R	L		R	L
15+	64	25.01±0.59	25.5±0.48	51	27.19±0.63	26.48±0.61
25+	66	26.06±0.49	25.5±0.46	44	26.72±0.67	25.60±0.61
35+	57	24.70±0.51	24.2±0.46	52	25.55±0.52	25.84±0.54
45+	46	25.07±0.71	25.1±0.71	41	26.30±0.67	25.26±0.75
55+	32	25.56±0.86	25.29±0.87	28	26.46±0.87	24.75±1.06
65+	29	24.77±0.54	24.81±0.78	32	25.12±0.72	24.67±0.64
75+	14	21.85±1.48	21.07±1.04	28	25.66±1.05	25.73±1.01

The mean forefoot angle for the right and left foot in females was 25.05^{o}±0.81 and 24.99±1.9 respectively, and for the males it was 26.20^{o}±1.03 and 25.47^{o}±0.3 respectively. These angles are larger than that generally accepted. Abnormally large forefoot angles were found in the right and left foot in females in 38.6% and 25.9% respectively, and in males in the right and left foot in 25.3% and 38.7% respectively. The correlation between hallux valgus of 35^{o}+ and increased forefoot angles was r=0.56 (female)and r=0.74 (males).

The data from this survey supports other reports which have stressed the relationship of increased metarsal angles and hallux valgus (Hardy and Clapham 1951). The evidence which relates intermetarsal angles to hallux valgus is in dispute. In children the valgus

changes have been noted before varus deviation of the 1st metatarsal (Hardy and Clapham 1951), while in another study comparing shoe wearing and non-shoe wearing Chinese, the subjects who did not wear shoes had less hallux valgus, but many more had increased inter-metarsal angles. Part of this may of course be related to the type of sandal worn by the Chinese (Sim-Fook and Hodgson 1958). No significant association between index minus or index plus-minus and hallux valgus was found. This is also a feature which other have commented upon (Hardy and Clapham 1951). The Namaqualand study while it shows an agreement between increased intermetarsal angles and hallux valgus, and between marked hallux valgus and increased forefoot angles, is incomplete. The aetiology of hallux valgus is much more complex than would be suggested by the simple measurements carried out on a radiograph (Piggot 1960). Hereditary plays a part (Johnston 1956) as does footwear (Sim-Fook and Hodgson 1958; Shine 1965) and pes planus. Nearly all the Namaqualand population wear shoes part of the time, from early adulthood, but no data is available on hereditary and other factors.

10.5. ARTICULAR HYPERMOBILITY

Articular hypermobility is a well accepted entity which resolves into several syndromes. In the absence of readily identified cause, the term hypermobility syndrome is used. It needs recognition clinically because it is a benign condition which causes joint pain and much anxiety (Kirk, Ansell and Bywaters 1967). The hereditary patterns of this syndrome have been discussed (Beighton and Horan 1970; Horan and Beighton 1971). More recently the benign attributes of the

syndrome have been challenged by the recognition of its association with mitral valve prolapse (Grahame, Edwards, Pitcher et al 1981) and with osteoarthrosis (Bird, Tribe and Bacon 1978). The evaluation of generalised hypermobility is still a cause for controversy but the scheme used in the study has proved useful in clinics.

Three hundred and twentyfive (325) adult males and 394 adult females and 537 children (268 male 269 female) were evaluated for articular hypermobility. Ninetyeight percent (98%) of males and 81% of females had articular hypermobility scores of 0 - 2, 1.53% of the adult males had scores larger than two, while 9.12% of the females had higher articular hypermobility scores. In the children there were higher hypermobility scores in males and females and for scores up to 6 there was little difference between the two groups, but 2.59% of the female children had scores 7 - 8. The data is demonstrated in the following Figure 10.5.1.

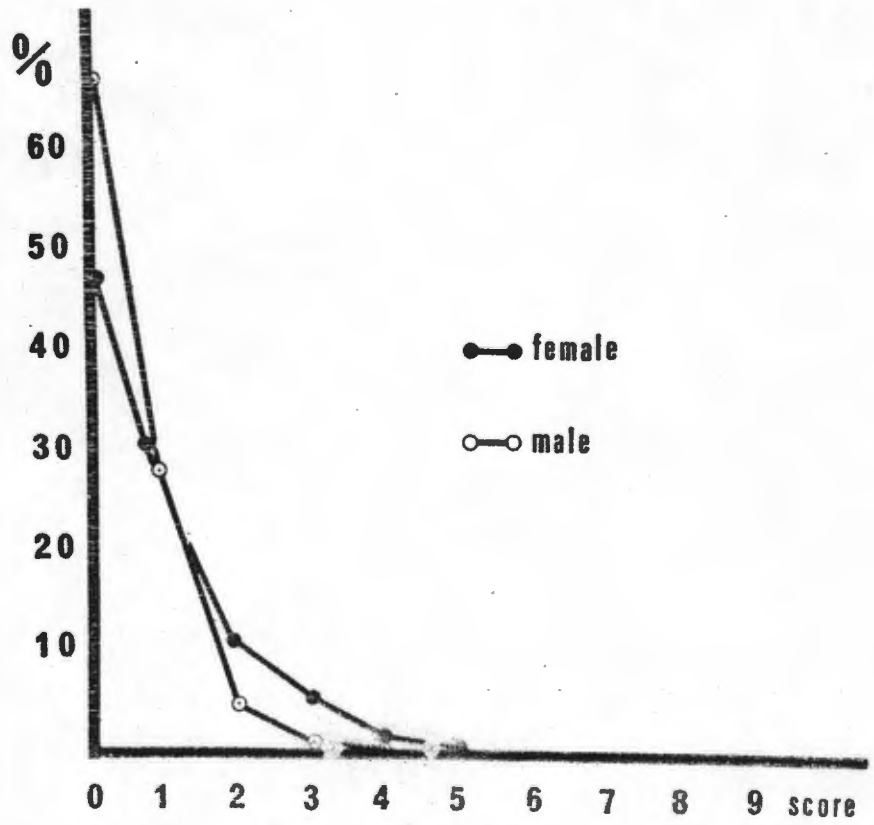


Fig10.5.1. ARTICULAR HYPERMOBILITY - ALL AGES

The data is similar to that reported for a Black rural population in the Transvaal (Beighton, Solomon and Soskolne(a) 1973). In a comparative study of joint laxity in White, Indian, Coloured and Black South Africans it was suggested that Indians had the greatest degree of joint laxity followed by Blacks, Coloureds and Whites (Schweitzer 1970). This study is difficult to evaluate because the author does not give details of the age distribution of the subjects in the universe which was studied.

10.6. OTHER ABNORMALITIES

Two subjects had a pes cavus for which no cause was found. One subject had a congenitally short 4th metacarpal, and one had a congenitally absent finger. There was one subject who had a ganglion on the dorsum of the hand, and one subject with a left hemiatrophy.

CHAPTER 11

AUTO-ANTIBODIES, SERUM COMPLEMENT AND SERUM URIC ACID

AUTO-ANTIBODIES

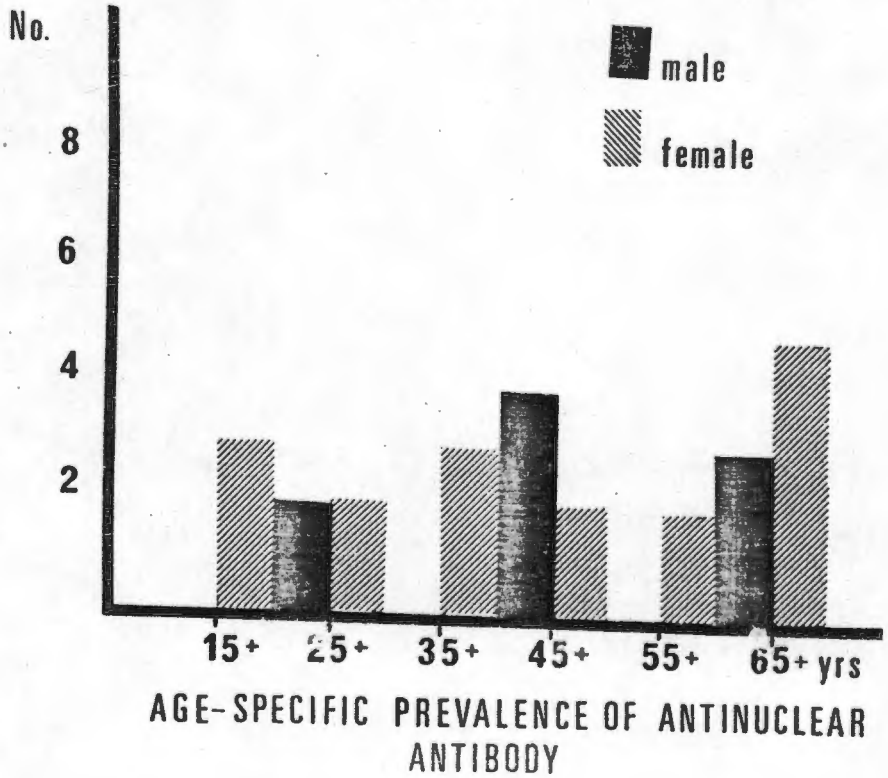
1.0. ANTIBODIES TO NUCLEAR CONSTITUENTS

1.1. Anti-nuclear Factor (ANF)

Anti-nuclear factor was measured in serum using a standard sandwich immunofluorescent technique, with rat liver sections as the substrate. All the sera were stored at - 20° until used. The sera were diluted 1:10 immediately prior to use. Positive sera were titred to an end point, and staining patterns were recorded. Twentysix (3.98%) of the 652 sera contained an anti-nuclear factor. The staining patterns were homogenous 17 (females 10, males 7) and speckled in 8 (females 7 and males 2).

The age specific distribution is shown in the following Figure 11.1.1.

Fig.11.1.1.



AGE-SPECIFIC PREVALENCE OF ANTINUCLEAR ANTIBODY

In females there was a peak of anti-nuclear factor in the 35+ age group (5.4%) followed by a decline and then a progressive rise to 9.4% in the 65+ age group. In the males there was a peak at the 45+ age group but no progressive increase with increasing age thereafter. The pooled data for the females/ males shows peaks at 45+ years and 65+ years. Most of the anti-nuclear factors were of low titre as shown in the following Table 11.1.1.

TABLE 11.1.1.

DISTRIBUTION OF THE TITRE OF ANTI-NUCLEAR FACTORS

		ANF TITRES				
	NO:	10	20	100	250	500
FEMALES	17	11	3	2	-	1
MALES	9	7	1	1	-	-

There were 17 subjects whose ANF showed a homogeneous staining pattern, and 8 in whom it was a speckled staining pattern. Only one subject whose anti-nuclear factor titre was 100 had possible SLE. One male aged 65 had rheumatoid arthritis and another male aged 28 years gave a history of polyarthritis and had both a positive latex and HEAT test, but this man showed no clinical or radiological evidence of active or inactive rheumatoid arthritis. One female aged 54 years with a speckled ANF to a titre of 10 gave a previous history of chest disease

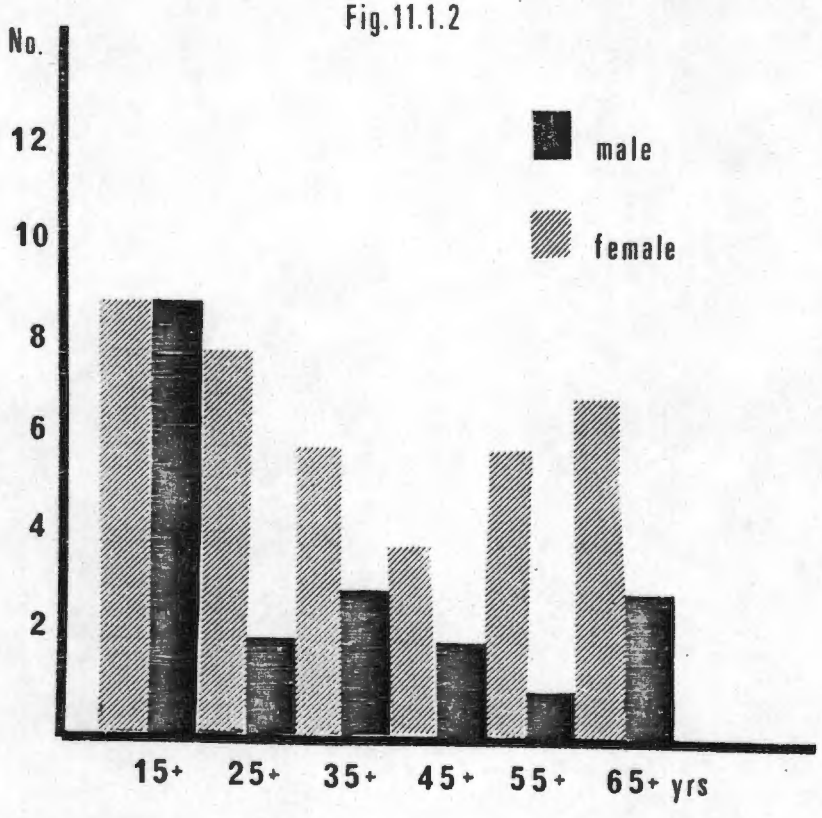
which required prolonged hospitalisation. It is presumed that this was an episode of pulmonary tuberculosis. None of the females were or had previously used contraceptive tablets. Anti-nuclear tests should always be evaluated in the light of the clinical picture. ANF is increased in the relatives of probands with systemic lupus erythematosus (Solheim and Larsen 1972) chronic infections such as tuberculosis (Friou 1967) in the ageing population (Cammarata, Rodnan and Fennell 1964) subjects with malignancy (Burnham 1972; Hamilton-Fairley 1972) and the drug treatment of arrhythmias, psychiatric disease, hypertension, epilepsy and tuberculosis (Zingale, Minzer, Rosenberg 1963; Fabius and Gaulhofer 1971; Dunstan, Taylor, Corcoran et al 1954; Kaplan, Wachtel, Czarnecki et al 1965). A fuller discussion of ANF and its relation to disease has been presented in an excellent review (Fernandez-Madrid, Mattioli 1976).

1.2. Extractable nuclear antigen

Antibody to extractable antigen (ENA) was measured using haemagglutination of human O cells. The sera were all screened at dilutions of 1:20 to 1:160, and all the positive sera (a 2+ reaction was regarded as the end point) of 1:80 or higher were titred to their end point. All the sera were also tested with counter-immunoelectrophoresis. One hundred and eightyseven of the female sera were positive, and 130 of the male were positive. The mean titre for the whole population was 66 $\pm$ 18.09. One hundred and eightyfive of the sera gave positive end points at

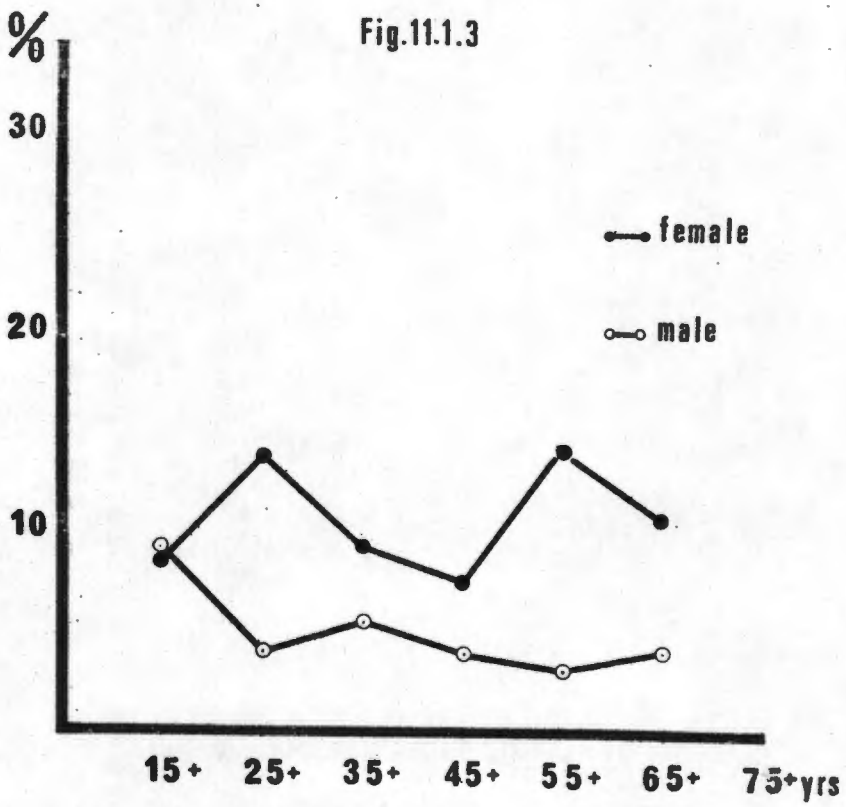
dilution of 20, 72 at dilution of 40, 33 at dilution of 80 and 29 at dilution of 160 or greater. The upper limit of normal for the population of two standard deviations is a dilution of 84. For the purposes of the analysis a titre of 80 was accepted as the upper limit of normal. Using this criterion the age distribution of anti ENA antibodies was as follows: (Figure 11.1.2. and Figure 11.1.3.).

Fig.11.1.2



DISTRIBUTION OF ANTI-ENA ANTIBODY

Fig.11.1.3



AGE-SPECIFIC PREVALENCE ANTI-ENA ANTIBODY

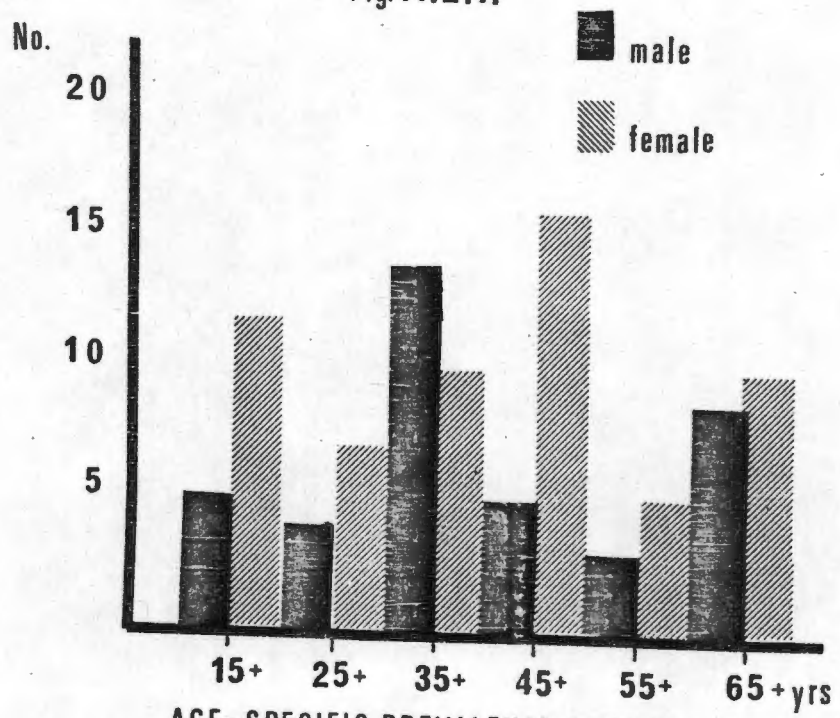
Haemagglutination titres of 1:80 were also considered positive for anti ENA antibody by other workers (McCain, Bell, Chodirker et al 1978). Thirtyseven sera (14 male, 23 female) were positive at titres of 80, twentytwo (6 male, 16 female) were positive at titres of 160, and five were positive at tires of 200 (2 male, 3 female). The ENA antibodies were further characterised to determine the number which were anti RNP antibodies. Thirtyone sera which were positive for anti ENA antibody were tested against RNAse treated ENA coated cells. Twelve sera were considered to have

an RNAse sensitive antibody (38%). None of the sera reacted positively with counter immuno-electrophoresis and very few were associated with positive anti-nuclear factors. The haemoglutination assay is a very sensitive test which is capable of detecting smaller antibody concentration than can be expected for the conventional immunofluorescent or counter immunoelectrophoresis tests. There is still not sufficient data to suggest that the RNAse insensitive sera are indeed all anti SM antibodies (Fernandez-Madrid and Mattioli 1976). Further studies will be needed to evaluate this assay. Of particular interest in this study is the relatively high prevalence of these antibodies, which do not show the well known rise with age which has been demonstrated for other auto-antibodies. Antibodies to RNP and SM (ENA)in high titre are associated with a variety of connective tissue syndromes notably mixed connective tissue disease and systemic lupus erythematosus (Barland 1975; Sharp, Irwin, May et al 1976) but no clinical examples were found in this study.

2.1. Smooth muscle antibody

Antismooth muscle antibody was the most commonly encountered antibody. One hundred sera were positive giving a prevalence of 15.3%. The distribution of this antibody in decades is given in the following Figure 11.2.1.

Fig.11.2.1.



AGE-SPECIFIC PREVALENCE OF SMOOTH MUSCLE ANTIBODY

The prevalence of this antibody peaked in the 35+ age group for males at 27.5% and in the 45+ age group of females at 34.0%. The titres of anti smooth muscle antibody were generally low as can be seen from the following Table 11.2.1.

TABLE 11.2.1.

DISTRIBUTION OF SMA TITRES IN MALES/FEMALES

		TITRES OF SMA				
	NO:	10	20	40	80	160
FEMALE	60	50	5	3	2	0
MALE	40	36	3	0	1	0

None of these subjects with positive anti smooth muscle antibodies showed evidence of overt chronic liver disease. The meaning of the anti SMA antibodies is not clear but its association with viral infection and malignancy are two explanations which may have relevance to the Namaqualand population (Holborow, Johnson, Farrow et al 1971; Holborow 1972).

3.1. Antigamma-globulin antibody (Rheumatoid factor)

IgM rheumatoid factor was measured in the serum using the Latex agglutination test and the human erythrocyte agglutination test (HEAT). All the sera for the Latex test were first screened with a diagnostic kit and the positive sera were titred using a conventional tube dilution method. A positive rheumatoid factor was found in 19 sera giving a prevalence of 2.9%. The age specific prevalence of rheumatoid factor (Latex and HEAT) is given in the following Table 11.3.1. for male and females.

TABLE 11.3.1.

AGE SPECIFIC PREVALENCE OF RHEUMATOID FACTOR

LATEX	15+	25+	35+	45+	55+	65+
MALES	1.6%	2.1%	2.0%	9.3%	0.0%	3.3%
FEMALES	0.0%	1.2%	0.0%	2.1%	2.9%	2.0%
TOTAL	0.70%	1.5%	1.0%	6.0%	2.0%	3.0%
HEAT						
MALES	0.0%	0.0%	2.0%	7.0%	0.0%	5.0%
FEMALES	0.0%	0.0%	0.0%	2.1%	2.9%	2.0%
TOTAL	0.0%	0.0%	1.0%	4.4%	2.0%	4.0%

There was a trend for an increasing prevalence of positive rheumatoid factor with age which was more clearly seen with the HEAT test than with the Latex test. The distribution of rheumatoid factor titre is demonstrated in the following Table 11.3.2.

TABLE 11.3.2.

DISTRIBUTION OF RHEUMATOID FACTOR TITRE

LATEX	NO:	40	80	160	320	640	1280	2560
MALES	12	3	1	2	1	2	3	0
FEMALES	6	2	0	0	1	2	1	0

The titre distribution for the HEAT test is given in the following Table 11.3.3.

TABLE 11.3.3.

DISTRIBUTION OF TITRES OF HEAT

HEAT	NO:	8	16	32	64	128	256
MALES	6	1	2	1	0	1	0
FEMALES	4	1	0	0	1	1	1

Three of the subjects with positive rheumatoid factor had rheumatoid arthritis by the New York criteria and one was regarded as a definite rheumatoid arthritis by the ARA criteria. In 6 subjects no cause for the positive rheumatoid factor was found wile in nine a possible cause was chronic obstructive lung disease in 1 previous exposure to silica in 1 and liver disease was suspected in 7 because of a reversed albumin/

globulin ratio, (one of these subjects also had a positive smooth muscle antibody) and a history of moderate too heavy alcohol consumptions.

4.1. Anti thyroid auto-antibodies

Anti-thyroid auto-antibodies were measured using thyroglobulin and thyroid microsomal antigen. The prevalence of anti-thyroglobulin antibody was 2.5% and for the anti-microsomal antibody it was 1.1%. The following Table 11.4.1. gives the age specific prevalence for males and females.

TABLE 11.4.1.

AGE SPECIFIC PREVALENCE OF THYROID AUTO ANTIBODIES

THYROGLOBULIN	15+	25+	35+	45+	55+	65+
MALES	3.2%	0.0%	0.0%	2.3%	3.4%	3.3%
FEMALES	5.4%	2.3%	1.8%	2.1%	2.9%	1.9%
TOTAL	5.1%	1.5%	0.9%	2.2%	3.2%	2.6%
MICROSOMAL						
MALES	0.0%	0.0%	0.0%	2.3%	0.0%	1.6%
FEMALES	2.7%	0.0%	0.0%	2.1%	2.9%	1.9%
TOTAL	1.7%	0.0%	0.0%	2.2%	1.6%	1.8%

No overt thyroid dysfunction was found in this population. One subject showed a reduced complement and complement factors suggesting a possible auto-immune disease. The titres of these two auto antibodies is shown in the following Table 11.4.2.

TABLE 11.4.2.

DISTRIBUTION OF THE TITRES OF ANTI-THYROGLOBULIN/ ANTI-MICROSOMAL ANTIBODIES

	10	20	40	80	160	320	640	1280	2560	5120
THYROGLOBULIN ANTIBODY	3	4	3	1	-	-	1	2	-	1
	100	400	1600	6400	25,600	102,000	409,600			
MICROSOMAL ANTIBODY	1	-	3	-	1	1	-			

The data on some of the auto antibodies shows the well-known increase with age which has been demonstrated repeatedly. It is however important to emphasise that the presence of these auto antibodies does not imply disease but their importance lies in the indication which they give of the presence in the body of a population of immunologically competent lymphocytes which are capable of mounting an aggressive immune response.

Comparison with other populations

Reports from other population studies show a measure of agreement with the Namaqualand data. Studies from Wales (Jacobs, Entwistle, Campbell et al 1969), New Zealand (Couchman, Wigley and Prior 1970), Australia (Whittingham, Irwin, McKay et al 1969; Hooper, Whittingham, Matthews et al 1972) have all shown a progressive rise in the prevalence of auto-antibodies with age and in one study half the population at age 60 had one or more auto-antibodies (Whittingham, Irwin, McKay et al 1969). The differences seen in the Namaqualand population are, a low prevalence of thyroid auto-antibodies with no

tendency to increase with increasing age. Rheumatoid factor also did not show a marked tendency to increase with increasing age. The difference between the Namaqualand population and other reported studies may be in their genetic background. All the reported studies have been on subjects of Caucasian extraction.

Mechanism of auto-immunity

The reason for the increase of auto antibodies with age have been discussed (Whittingham, Irwin, McKay et al 1969). Several theories have been suggested. Firstly it may be that as people age they have a greater chance of meeting the hypothetical triggering factor. Secondly it has been suggested that with age there is increasing structural protein changes which can provoke the immune response (non-self antigen). This has been shown to occur in collagen (Verzar 1957) and aortic elastin (Labella and Lindsay 1963; Ram 1967). The third possibility is that the antibodies represent subclinical auto-immune disease, but there is not much evidence to favour this idea. Rheumatoid factor has been shown to protect against certain viral infections, while a clearing mechanism for tissue antigens have been claimed for some naturally occuring auto-antibodies. There is too, some evidence that this hyper-immune reaction could have other benefits, for instance in the surveillance against malignant cells. Part of the evidence for this comes from the observation that the incidence of malignancy is lower in those with an allergic diathesis (Fisherman 1960; MacKay 1966).

4.1.1. Serum complement

The serum total haemolytic complement profile in this community is shown in the following Table 11.4.3.

TABLE 11.4.3.

DISTRIBUTION OF TOTAL HAEMOLYTIC COMPLEMENT IN MALES AND FEMALES (UNITS/ML.)

COMPLEMENT LEVEL	MALES	FEMALES
50 units/ml.	14	15
51-99 " "	17	21
100-149 " "	61	72
150-199 " "	147	172
200-249 " "	52	58
250+ " "	8	15

The distribution of complement levels is not significantly different in males and females. All those sera with levels under 50 units/ml. were tested for their C3 and C4 content. Three had low C3 levels, and three had low C4 levels which may reflect classic complement pathway activation but there was no clinically obvious disease.

5.1. Serum Uric Acid

Clinical gout was not encountered in the population and there was no radiological evidence to suggest tophaceous gout. The absence of gout from this population is hardly surprising in view of the low serum uric acid levels which were found. Acute gout is generally the consequence of hyperuricaemia and the levels of the hyperuricaemia

have been shown to determine the incidence of acute gouty arthritis (Hall, Barry, Dawber et al 1967). The distribution of the mean age specific serum uric acids for males and females is shown in the following Table 11.5.1. and Table 11.5.2.

TABLE 11.5.1.

MEAN, AGE SPECIFIC SERUM URIC ACID - MALES

AGE GROUP	NO: EXAMINED	MEAN SEM
15 - 24	62	0.234 ± 0.006
25 - 34	48	0.237 ± 0.009
35 - 44	47	0.237 ± 0.008
45 - 54	45	0.255 ± 0.009
55 - 64	28	0.265 ± 0.013
65 - 74	29	0.251 ± 0.012
75+	28	0.264 ± 0.016

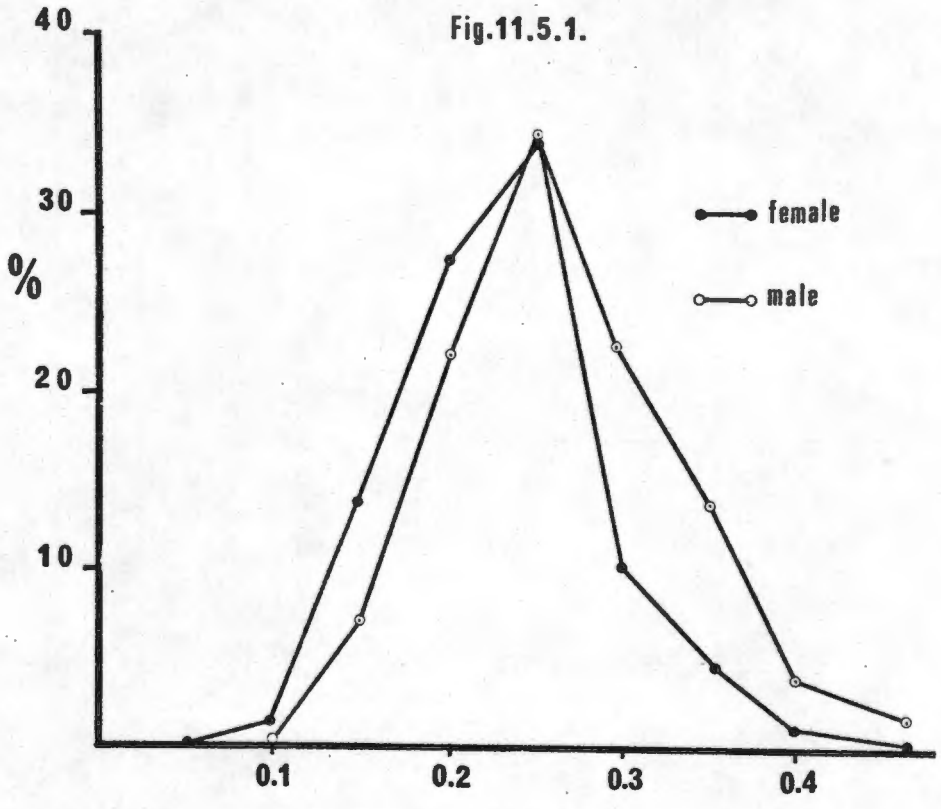
TABLE 11.5.2.

MEAN AGE - SPECIFIC SERUM URIC ACID - FEMALES

AGE GROUP	NO: EXAMINED	MEAN SEM
15 - 24	74	0.212 ± 0.008
25 - 34	80	0.202 ± 0.006
35 - 44	54	0.199 ± 0.007
45 - 54	46	0.207 ± 0.013
55 - 64	31	0.239 ± 0.013
65 - 74	34	0.246 ± 0.009
75+	17	0.245 ± 0.018

The concentrations of serum uric acid show that in males there was a slight rise reaching a peak at 55 - 64 years, whereas in women the concentrations remain lower than in males up to the age of 55 when the serum uric acid rose to levels which were only slightly less than the mean concentrate in the males. The frequency distribution of serum uric acid is demonstrated in the following Figure 11.5.1.

Fig.11.5.1.



FREQUENCY DISTRIBUTION OF SERUM URIC ACID

The curves show the well-known characteristic Unimodal curve with a shift to the left for the females. There was a poor correlation

between serum uric acid and haemoglobin levels for females up to 40 years (r= 0.40 > p= 0.05) and for those over 45 years (R= 0.18 p= > 0.05). In the males the correlation between normal haemoglobin values and serum uric acid was r= 0.36 (p= < 0.1 > 0.05). The correlation between body mass and serum uric acid and lean body mass and serum uric acid was r= 0.32 (p= ns) and r=0 .66 (p=<0.01 > 0.001) respectively for males and r=0.18 (p= ns) and r=0 .49 (p=< 0.05 > 0.02) resepctively for females. Others have shown that there is a correlation between obesity and serum uric acid (Nichols and Scott 1972; Acheson and Chan 1969) but cogent arguments have been advanced which suggest that it is not adiposity but muscle bulk which determines the level of uric acid (Fessel and Barr 1977) and the data from this population study supports the suggestion that it is the lean body mass which correlates with serum uric acid.

The lack of changes in mean serum uric acid with age in adult males and the rise in females with age has been demonstrated before, and the reasons for the sex difference with age have been discussed (Mikkelsen, Dodge, Valkenberg et al 1965). It has been shown that stilboestrol will reduce serum uric acid by enhancing water clearances in men, and that urate clearances are higher in women than in men (Nichols, Snaith, Yablonsky et al 1973). It also seems that of the several factors which influence the serum uric acid, it is at least partially dependant on Haemoglobin (Acheson and O'Brien 1966). The association is stronger for men than for women. This was not demonstrated in the Rietpoort study. The dependance on haemoglobin

presumably reflects the rate at which red blood cells are destroyed. These are two facts which do offer some reasons for the differences of serum uric acid between males and females. According to this data, the rise in serum uric acid after the menopause is considered to result from decreased oestrogen production and the cessation of red blood cell loss with the menses.

The concentrations of mean serum uric acid in this study is lower than that of other studies. The differences in serum uric acid which have been reported (Prior, Rose and Davidson 1969; Burch, O'Brien, Need et al 1966; Prior, Rose, Harvey et al 1966; Beighton, Solomon, Soskolne and Sweet 1973) is partly a reflection of ethnic differences but environment appears to play an important role as well (Rose and Prior 1963). Thus there are significant differences in serum uric acid between rural and urban South African Blacks (Beighton, Solomon, Soskolne and Sweet 1973; Beighton, Solomon, Soskolne, Sweet and Robin 1974). The shift from a rural to an urban way of life is associated with a change in eating habits, social class, access to medication such as aspirin. The change also exposes the individual to stress, but how this would operate is not clear. In the Namaqualand population it is clear that most of the people were lean and their socio-ecconomic circumstances would not have allowed a diet rich in protein/lipid. Most of the males took alcohol, but the pattern of heavy weekend drinking, which is common, differs from the regular alcohol intake which appears to be characteristic of some patients with gout (Grahame and Scott 1970). There is an association between gout and hypertension, but not in non-gouty

hyperuricaemia. This is bourne out to some extent by this study where serum uric acid levels tended to remain low in spite of a prevalence of hypertension of 13%.

6.0. Serological tests for syphilis

The sera of 40 females (11.4%) and 22 (7.48%) of the males gave positive reactions in the VDRL reaction. The overall prevalence of a positive VDRL reaction was 9.62%. Positive TPHA reactions were present in 95% of the female and 90% of the male sera which had a positive VDRL. One subject had secondary lues, and another had a palatal perforation which was presumed to be a healed guamma.

CHAPTER 12

ILLNESS PROFILE

ILLNESS PROFILE

Although rheumatic disease in this Namaqualand population forms the basis of this thesis, it is relevant to give it some perspective. The study was not designed for a total illness profile but some information was obtained from the questionnaire and the clinical examination which gives an idea of the illness profile of the population.

The population receives its medical care from general practitioners in the neighbouring towns, but many consult the sister in charge of the Mission hospital. She is very competent and well-trained and she diagnoses and prescribes for simple medical conditions. She is supported by a general practitioner, who is the District Surgeon. He consults once a month and his expertise provides another tier of medical diagnosis and treatment. Serious illness is generally referred to the nearest hospital at Vredendal or to the two teaching hospitals in Cape Town.

The service which the Mission hospital provides is private and those who cannot afford medication are subsidised by the church. Old age pensioners and those on disability grants receive free medical attention from the district surgeon. The financial constraints on the church and the great distance which individuals have to travel make it difficult or impossible to provide adequate long term care.

The hospital register of Rietpoort

To obtain an idea of the illness profile of this community it was

decided to examine the hospital register for two periods, 1969 and 1978 and the medical diagnoses which were recorded by the Sister for 1969 and 1978 were analysed. Neither an accurate diagnosis nor a strict taxonomy can be expected from such a simple medical facility. It was nevertheless possible to examine disease categories according to systems involved because this seemed to provide at least some idea of the nature and the extent of disease in this community. The type of illness recorded is shown in the following Table 12.1.

TABLE 12.1.

MEDICAL DIAGNOSES IN 1969 and 1978

DIAGNOSES	1969		1978	
	NO:	%	NO:	%
Respiratory disease	518	26.9	773	26.2
Skin disease	469	24.3	802	17.4
Cardiac/Blood pressure	45	2.3	70	2.4
E.N.T. disease	254	13.2	299	10.2
Gastro-intestinal disease	176	9.1	258	8.8
Obstetrics	125	6.5	234	7.8
Immunisations	89	4.6	60	2.0
Trauma	58	3.0	77	2.6
Neurological disease	39	2.0	27	0.9
Musculo-skeletal	32	1.6	72	2.5
Venereal disease	30	1.6	38	1.3
Genitourinary disease	22	1.1	51	1.7
Anaemia	14	0.7	77	2.6
Undiagnosed	56	2.9	91	3.1
	1927		2929	

The pattern of illness over the 9 year period showed some minor changes. The major bulk of medical problems seen at the hospital was shared by skin and respiratory disease. In both of these categories skin and respiratory infections were the most common. Cardiovascular disease was not commonly recorded but this differs from the data on hypertension found during this survey (see page 174). This is not surprising since hypertension is not symptomatic until organ failure occurs. The involvement of the musculo-skeletal system likewise is not recorded frequently. There is a well-known disparity between the prevalence of rheumatic complaints and the frequency with which sufferers seek medical attention. Thus in the town of Leigh, 50% of women who had symptomatic osteoarthrosis, and 60% of those with disc disorders did not seek medical attention (Kellgren, Lawrence and Aitken-Swan 1953). The self-limiting and/or relapsing - remitting nature of many of the rheumatic diseases can be expected to contribute in large measure to the disparities mentioned. What effect an all pervading gloomy outlook about treatment has in determining visits to medical services is unknown for this population (Wood 1977).

Medical problems found during the survey

The types of medical problems encountered during this survey are shown in Table 12.2. The diagnoses were again grouped into broad systems rather than itemising small numbers of individual diseases.

TABLE 12.1.

ILLNESS FOUND IN MALES AND FEMALES IN NAMAQUALAND (734 INDIVIDUALS)

DISEASE	15+	25+	25+	45+	55+	65+	TOTAL
SKIN	20	1	12	1	-	17	51
RESPIRATORY	-	1	2	-	1	1	5
NEUROLOGICAL	-	1	-	4	1	5	11
PSYCHIATRIC	-	3	1	1	1	1	7
CARDIOVASCULAR	5	7	12	18	20	35	96
NEOPLASMS	-	-	-	-	1	3	4
VENEREAL DISEASE	-	1	1	-	-	-	2
							176

Cardiovascular disease was the most frequently recorded diagnosis. Hypertension made up the over-whelming majority of diagnoses in this category (13% of the population) followed by skin disease (6.9%) and neurological disease (1.5%). The skin diseases were varied and ranged from eczema to sun damage, discoid lupus erythematosus, alopecia and psoriasis. There were 3 subjects with a hemiplegia (0.4%), one with a spastic paraparesis and one with Parkinsons disease. The four subjects with neoplasms were all previously diagnosed cancers of the breast and stomach.

Hypertension in the Rietpoort population

The following Table 12.3. analyses the hypertension encountered in this population:

TABLE 12.3.

THE PREVALENCE OF HYPERTENSION IN MALES AND FEMALES IN NAMAQUALAND - (Diastolic over 100 mm Hg)

	15+	25+	35+	45+	55+	65+
MALES	1.1%	1.1%	8.2%	13.6%	15.2%	9.4%
FEMALES	3.7%	7.6%	12.9%	23.5%	24.4%	40.9%

It can be seen that the prevalence of hypertension increased progressively in females from 3.7% in those aged 15 - 24 to 40.9% in those over 65 years. The prevalence of hypertension in males was much less, and it peaked in the 55 - 64 year group.

The degree of diastolic hypertension is shown in the following Table 12.4.

TABLE 12.4.

AGE SPECIFIC PREVALENCE OF DIASTOLIC HYPERTENSION IN MALES AND FEMALES IN NAMAQUALAND

DIASTOLIC PRESSURE	SEX	15+	25+	35+	45+	55+	65+
100 - 109 mm Hg	M	-	1.1%	6.1%	6.8%	9.0%	5.6%
	F	0.9%	1.3%	4.8%	13.7%	21.9%	22.9%
110 - 119 mm Hg	M		-	-	2.3%	3.0%	3.7%
	F	2.8%	6.4%	6.4%	3.9%	9.8%	11.5%
120+ mm Hg	M	-	-	2.0%	6.8%	6.0%	3.7%
	F	-	-	1.6%	5.8%	4.8%	6.5%

The data shows that the progressive increase in the prevalence of hypertension is a feature of milder diastolic hypertension and that it is similar in males and females up to the age of 45 years. Thereafter there is a steep rise in the prevalence in females. For the more severe grades of diastolic hypertension females and males were almost equally affected. The prevalence of hypertension in Whites, Coloureds and Blacks exists. In rural Blacks the prevalence is low (Seedat, Hackland and Mpontshane 1981) but it is higher in urban Blacks (Seedat and Reddy 1974, Seftel, Johnson and Muller 1980). In Whites the prevalence is lower (Seedat, Seedat and Veale 1980), while in Coloured South Africans the prevalence of hypertension has not been studied in strict epidemiological terms. There is some evidence that it is intermediate between White and Black South Africans, but this is based on a hospital record and the data is 25 years old (Schrire 1958).

CONCLUSION

CONCLUSION

Epidemiology, the pathology of families, communities or large groups, sets out:

(1) To count the number of individuals affected by a disease

(2) To relate the occurrence of a particular disease or attribute in similar individuals under different conditions

(3) To provide a measure of disease frequency, its personal and community effects and in this way it is the 'afferent' limb of health care provision

The study of the population of Rietpoort has fulfilled in part the first function of epidemiology in so far as it has measured the prevalence rate of the following rheumatic diseases:

1.	Osteoarthrosis - all grades of severity	420/1000
	- severe osteoarthrosis	119/1000
2.	Soft Tissue Rheumatism	86/1000
	Shoulder tendinitis	69/1000
	Lateral epicondylitis	16/1000
	Carpal tunnel syndrome	3/1000
	Bursitis (olecranon)	3/1000
	De Quervains tenosynovitis	3/1000
	Dupuytren's contracture	10/1000
	Fibromyalgic syndrome	3/1000
3.	Osteoporosis - all ages	30/1000
	- subjects over 65 years	126/1000
4.	Rheumatoid arthritis	4/1000

This study was not concerned directly with the second function of epidemiology but it does provide a start to the study of rheumatic disease in Coloured South Africans. It will hopefully provide a useful basis for comparison with other studies.

The third function of epidemiology has not been developed in this study. A study of the impact of disease at a personal and community level will require a different approach. In some sense the Rietpoort study has tentatively started to point this way because it

has provided an insight into the rheumatic problems of a rurally living community, the effects of which can only be guessed at present. It may be that the community of Rietpoort is insignificant in global South African terms, but is is a microcosm and what has been studied there has relevance to the whole. I believe that it tells us that there is a need to provide better health care for isolated communities and that it should encourage us to look at rheumatic disease and rheumatological services in South Africa as an important priority. As a teacher concerned with under and postgraduate rheumatology education, the study has provided much to think about. It has changed the content and the philosophy of rheumatology teaching which is perhaps where the provision of better rheumatology services should start.

APPENDIX

NAME: NO:

ADDRESS: DATE OF BIRTH

HISTORY:

1. How long have you lived in this area?
2. Where else have you lived?
   (give number of years if possible)
3. Which school did you attend?
4. Where have you worked previously?
5. Have you had any serious illnesses?
6. Occupation?
7. What is the family income?

CHEST SURVEY:

8. Have you ever suffered from asthma?
9. Had a chronic cough?
10. Coughed up mucous over a long time?
11. Been short of breath?
12. Suffered from Tuberculosis?
13. Been hospitalised for chest trouble?

DO YOU SMOKE?

14. Have you heard that smoking is harmful?
15. How long have you smoked?
16. How much do you smoke?
17. Why did you start smoking?
    1. Because you liked it?
    2. Because you wanted to be more adult?
    3. Because friends smoked?
    4. Don't know?
18. Do you use an open fire in your house?
19. How many people live in the house?

20. Have you had or do you have backache?

    Duration longer than 3 months

    Insidious/sudden onset

    Is the back stiff in the mornings?

    Does it improve with exercise

21. Have your joints ever swelled up?

    When?

    How many swelled up?

    How long did this last?

    Are your joints stiff in the morning?

22. Have you had/do you have pain in shoulders?

23. Does your skin burn easily in the sun?

24. Do your fingers change colour in cold weather

25. Have you noticed your hair becoming thinned?

26. Have you ever had pleurisy, chest pain?

27. Do you or did you ever have fits/mental problems?

28. Have you taken contraceptive pills?

EXAMINATION:

Height standing Height sitting Weight

Skinfold thickness B.P.

	Yes	No
1. Facial erythema, pigmentation		
2. Discoid L.E.		
3. Thinning or "frontal fracturing" of hair		
4. Oral/nasopharyngeal ulcers, (not aphthous)		
5. Pleural/pericardial rub		
6. Arthritis - soft tissue swelling, warmth		
- nodules		
- which joints?		
7. Psoriasis - skin, nails, scalp		
8. Other skin lesions		

	Right	Left
9. Hypermobility Little finger to 90°		
Thumb to forearm		
Elbow extension 10°		
Knee extension 10°		
Forward bending - palms on floor		
10. Tennis elbow - pain on resisted flexion of wrist (elbow extended)		
11. Shoulder mobility : Flexion 0 - 90°		
Extension		
Internal rotation		
Abduction 0 - 180°		
12. Spinal mobility : Flexion		
Extension		
Lateral bending		

	Yes	No
13. Arthritis		
Soft tissue swelling of a joint		
Swelling in another joint		
Symmetrical joint swelling		
Is there swelling/limitation ROM or subluxation of 3 joints?		
Is there a hand/foot involved? (Irreducible subluxation of MTPs)		

MCP OSTEOARTHROSIS : GRADE 3 ON THE RIGHT/GRADE 2 ON THE LEFT

MCP OSTEOARTHROSIS : GRADE 3 (NOTE THE RELATIVE SPARING OF THE THUMB)

MCP OSTEOARTHROSIS (GRADE 4 RIGHT, GRADE 3 LEFT) THE MIDDLE FINGER MCP IS THE WORST AFFECTED

MCP OSTEOARTHROSIS SEVERE DISEASE (GRADE 4)

A. TABLE 7A

NUMBER OF AFFECTED TIP JOINTS (GRADE 2 - 4) IN MALES

AGE		GRADE 2				GRADE 3				GRADE 4			
		1	2	3	4	1	2	3	4	1	2	3	4
15+	R	1	0	1	1	0	1	0	0	0	0	0	0
	L	1	0	1	1	0	1	0	0	0	0	0	0
25+	R	0	0	0	0	0	0	0	0	0	0	0	1
	L	0	0	0	2	0	0	0	0	0	0	0	1
35+	R	4	4	4	6	0	0	2	1	0	0	0	0
	L	6	2	5	7	0	0	0	0	0	0	0	0
45+	R	3	6	3	6	1	1	0	2	0	1	0	0
	L	2	5	5	6	1	1	0	1	0	0	0	0
55+	R	7	6	7	10	0	5	2	0	0	0	0	0
	L	5	7	4	9	0	4	1	2	0	0	0	0
65+	R	17	18	9	17	9	11	12	7	2	3	1	1
	L	16	12	14	22	5	7	2	7	3	4	0	2

A. TABLE 7B

NUMBER OF AFFECTED TIP JOINTS (GRADE 2 - 4) IN FEMALES

AGE		GRADE 2				GRADE 3				GRADE 4			
		1	2	3	4	1	2	3	4	1	2	3	4
15+	R	0	0	1	1	0	0	0	0	0	0	0	0
	L	0	0	0	0	0	0	0	0	0	0	0	0
25+	R	0	0	0	0	0	0	0	1	0	0	0	0
	L	0	1	0	0	0	0	0	0	0	0	0	0
35+	R	1	2	2	0	1	0	0	0	0	0	0	0
	L	1	1	1	0	0	0	0	0	0	0	0	0
45+	R	7	6	4	9	1	1	0	2	0	0	1	0
	L	5	5	5	7	0	1	0	0	0	0	0	0
55+	R	10	10	6	10	1	2	3	5	0	0	1	0
	L	4	7	6	7	1	2	0	3	0	0	1	0
65+	R	10	15	10	14	5	4	3	6	1	1	1	2
	L	9	11	7	12	5	2	0	8	0	1	2	0

A. TABLE 7C

THE NUMBER OF AFFECTED PIP JOINTS (GRADE 2 - 4) IN MALES

AGE		GRADE 2					GRADE 3					GRADE 4				
		1	2	3	4	5	1	2	3	4	5	1	2	3	4	5
15+	R	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	L	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
25+	R	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	L	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0
35+	R	6	3	3	0	2	0	0	0	0	0	0	0	0	0	0
	L	4	1	2	1	2	0	0	0	0	0	0	0	0	0	0
45+	R	6	4	1	1	2	1	0	1	0	0	0	0	0	0	0
	L	9	3	0	0	1	0	0	0	0	0	0	0	0	0	0
55+	R	8	5	4	3	4	2	0	2	1	2	0	0	0	0	0
	L	10	4	6	3	3	2	0	2	1	2	0	0	0	0	0
65+	R	15	12	17	8	15	16	4	3	2	2	0	0	0	2	0
	L	16	11	14	8	17	12	3	3	3	3	1	0	0	0	0

A. TABLE 7D

THE NUMBER OF AFFECTED PIP JOINTS (GRADE 2 - 4) IN FEMALES

AGE		GRADE 2					GRADE 3					GRADE 4					
		1	2	3	4	5	1	2	3	4	5	1	2	3	4	5	
15+(65)	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	R
	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	L
25(54)	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	R
	0	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	L
35(52)	2	2	2	0	2	0	0	0	0	0	0	0	0	0	0	0	R
	1	1	2	0	0	1	0	0	0	0	0	0	0	0	0	0	L
45(43)	10	5	2	1	4	1	0	0	0	1	0	0	0	1	0		R
	9	3	3	0	1	0	0	0	0	1	1	0	0	0	0		L
55(31)	28	13	11	7	10	7	1	2	3	2	1	0	0	0	2		R
	27	10	9	6	14	6	1	3	2	0	1	0	0	0	1		L
65(42)	14	10	6	4	9	12	1	2	3	3	1	0	0	0	0		R
	16	11	11	3	11	10	1	2	3	1	0	0	1	0	0		L

A. TABLE 7E

AGE SPECIFIC PREVALENCE OF OSTEOARTHROSIS OF PIP JOINTS

	MALE					FEMALE			
	ALL GRADES		GRADE 3 - 4			ALL GRADES		GRADE 3 - 4	
15+ (51)	1	0.98%	0	0%	(65)	0	%	0	%
25+ (45)	2	2.22%	1	1%	(54)	2	1.85%	2	0.92%
35+ (46)	10	10.86%	0	0%	(52)	5	4.80%	0	-
45+ (29)	11	18.96%	2	3.44%	(43)	15	17.44%	2	2.32%
55+ (24)	15	31.25%	9	18.75%	(31)	23	37.09	5	8.06%
65+ (61)	45	36.88%	24	19.67%	(42)	29	34.52%	16	19.04%
256					287				

Overall % = 84/512 = 16.4%

Over 35+ = 25.31%

Overall = 74/574 = 12.89%

Over 35+ = 72/336 = 21.42%

OVERALL = 158/1086 = 14.54%

OVER 35+ 153/656 = 23.32%

A. TABLE 7F

AGE SPECIFIC PREVALENCE OF GRADE 2 OA OF THE 1ST - 5TH MCP JOINT

MALES

	1ST		2ND		3RD		4TH		5TH	
	R	L	R	L	R	L	R	L	R	L
15+	1.9	1.9	0.0	0.0	1.9	1.9	0.0	0.0	0.0	0.0
25+	2.2	2.2	0.0	0.0	2.2	2.2	0.0	0.0	0.0	0.0
35+	2.1	4.3	4.3	4.3	4.3	6.5	4.3	0.0	2.1	0.0
45+	6.8	6.8	3.4	3.4	6.8	3.4	6.8	3.4	0.0	0.0
55+	8.3	8.3	16.6	8.3	12.5	4.1	4.1	4.1	4.1	0.0
65+	13.1	13.1	6.5	3.2	4.9	0.0	9.8	3.2	4.9	1.6

A. TABLE 7G

AGE SPECIFIC PREVALENCE OF GRADE 3 OA OF METACARPOPHALANGEAL JOINTS

IN MALES - RIGHT HAND AND LEFT HAND

	1ST		2ND		3RD		4TH		5TH	
	R	L	R	L	R	L	R	L	R	L
15+	0.0	0.0	1.9	1.9	0.0	0.0	0.0	0.0	0.0	0.0
25+	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
35+	4.3	0.0	0.0	0.0	0.0	0.0	2.1	0.0	0.0	0.0
45+	6.8	0.0	6.8	3.4	6.6	3.4	0.0	0.0	0.0	0.0
55+	20.8	16.6	4.1	8.3	16.6	8.3	0.0	0.0	0.0	0.0
65+	19.6	19.6	8.1	9.8	18.0	0.0	3.2	1.6	0.0	0.0

AGE SPECIFIC PREVALENCE FOR GRADE 4 OA OF 1ST-5TH MCP JOINTS IN MALES

	1ST		2ND		3RD		4TH		5TH	
	R	L	R	L	R	L	R	L	R	L
15+	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
25+	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
35+	2.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
45+	0.0	0.0	0.0	0.0	0.0	3.4	0.0	0.0	0.0	0.0
55+	4.1	3.4	8.3	8.3	8.3	8.3	0.0	0.0	0.0	0.0
65+	4.9	3.2	9.8	3.2	9.8	0.0	1.6	0.0	0.0	0.0

A. TABLE 7H

METACARPOPHALANGEAL OA (GRADE 2 - 4) IN FEMALES

AGE		GRADE 2					GRADE 3					GRADE 4				
		1	2	3	4	5	1	2	3	4	5	1	2	3	4	5
15+	R	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0
	L	0	0	0	0	0	0	0	1	0	0	0	1	0	0	0
25+	R	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0
	L	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0
35+	R	2	3	3	1	1	0	0	0	0	0	0	0	0	0	0
	L	1	2	3	0	1	0	0	0	0	0	0	0	0	0	0
45+	R	2	0	1	0	1	0	0	0	0	0	0	0	0	0	0
	L	1	1	1	0	2	0	0	0	0	0	0	0	0	0	0
55+	R	2	0	3	1	1	4	1	1	0	0	1	0	0	0	0
	L	2	0	0	1	1	2	0	0	0	0	0	0	0	0	0
65+	R	5	5	3	1	0	3	1	3	0	0	0	1	2	0	0
	L	5	5	4	2	1	1	2	3	0	0	0	0	1	0	0

A. TABLE 7J

AGE SPECIFIC PREVALENCE OF OSTEOARTHROSIS OF THE MCP JOINTS

	MALE					FEMALE			
	ALL GRADES		GRADE 3 - 4			ALL GRADES		GRADE 3 - 4	
15+ (51)	2	1.96%	1	0.98%	(64)	0	-	0	-
25+ (45)	1	1.11%	0	-	(54)	2	1.85%	0	-
35+ (46)	7	7.6%	4	4.34%	(52)	5	4.80%	0	-
45+ (29)	5	8.62%	3	5.17%	(43)	15	17.44%	2	9.67%
55+ (24)	12	25.00%	10	20.83%	(31)	10	16.12%	6	5.35%
65+ (61)	34	27.86%	27	22.13%	(42)	15	17.8%	9	10.71%
256					287				

Overall = 61/512 = 11.91%

35+ = 58/320 = 18.12%

Overall = 47/574 = 8.18%

Over 35 = 45/336 =13.39%

OVERALL = 108/1086 = 9.94%

OVER 35+= 103/656 =15.70%

A. TABLE 7K

CARPOMETACARPAL OSTEOARTHROSIS IN MALES/FEMALES

AGE	GRADE 2	GRADE 3	GRADE 4
15+ Rm	2	0	0
Lm	2	0	0
15+ Rf	0	0	0
Lf	0	0	1
25+ Rm	4	0	0
Lm	3	1	0
Rf	1	0	0
Lf	1	0	0
35+ Rm	3	0	0
Lm	3	0	0
Rf	5	0	0
Rf	5	0	0
45+ Rm	4	1	0
Lm	5	0	0
Rf	7	1	0
Lf	6	2	0
55+ Rm	5	0	0
Lm	5	1	0
Rf	6	0	1
Lf	10	0	1
65+ Rm	18	3	2
Lm	17	4	0
Rf	11	8	1
Lf	12	7	0

A.10.4.2.

AGE SPECIFIC INTERMETARSAL ANGLES IN MALES AND FEMALES (GREATER THAN NORMAL*)

	FEMALES					MALES				
AGE	NO	R		L		NO	R		L	
15+	64	27	42.2%	25	39.1%	51	18	35.3%	20	39.2%
25+	66	28	42.4%	23	34.8%	44	15	34.1%	13	29.5%
35+	57	18	31.5%	15	26.3%	52	14	26.9%	18	34.6%
45+	46	15	32.6%	10	21.7%	41	7	17.1%	15	36.5%
55+	32	10	31.3%	12	37.5%	28	6	21.4%	3	10.7%
65+	29	11	37.9%	10	34.5%	32	6	18.75%	7	21.9%
75+	14	7	50.0%	3	21.4%	28	7	25.0%	8	28.5%
	308	116		98		276	73		84	

* Mean intermetarsal angle Females Rt foot $8.90 \pm 0.51 \times 1.96 = 7.91 - 9.89^{o}$

Lft " $8.90 \pm 0.65 \times 1.96 = 7.63 - 10.17^{o}$

Males Rt foot $8.25 \pm 0.35 \times 1.96 = 7.57 - 8.99^{o}$

Lft " $8.27 \pm 0.37 \times 1.96 = 7.55 - 8.99^{o}$

A.10.4.3.

ABNORMALLY LARGE FOREFOOT ANGLES IN MALES AND FEMALES

	FEMALES					MALES				
AGE	NO	R		L		NO	R		L	
15+	64	24	37.5%	19	29.6%	51	17	33.3%	25	49.0%
25+	66	29	43.9%	20	30.3%	44	12	27.3%	14	31.8%
35+	57	20	35.1%	11	19.2%	52	11	21.2%	18	34.6%
45+	46	22	47.8%	10	21.7%	41	7	17.0%	16	39.0%
55+	32	11	34.4%	11	34.4%	28	9	32.1%	13	46.4%
65+	29	10	34.5%	8	27.5%	32	6	18.8%	11	34.4%
75+	14	3	21.4%	1	7.1%	28	8	28.5%	10	35.7%
	308	119		80		276	70		107	

Mean Forefoot angle Female R = $25.0^{o} \pm 0.81 \times 1.96$ = Range $23.4^{o} - 26.6^{o}$

" L = $24.9^{o} \pm 1.9 \times 1.96$ = " $21.2^{o} - 28.6^{o}$

Mean Forefoot angle Male R = $26.2 \pm 1.03 \times 1.96$ = Range $24.2^{o} - 28.2^{o}$

L = $25.5 \pm 0.30 \times 1.96$ = " 24.92 - 26.10

BIBLIOGRAPHY

Abdin, Z.H. (1971). Benign polyarthritis in Egyptian children and youth. Abstr. 23 presented at 7th European Congress of Rheumatology.

Acheson, R.M., Chan, Y.K. (1969). New Haven Survey of joint diseases. Prediction of serum uric acid in a general population. J.Chronic Dis. 21:543.

Acheson, R.M., O'Brien, W.M. (1966). Dependence of serum uric acid in haemoglobin and other factors in the general population. Lancet 2:777.

Adler, E., Abramson, J.H., Elkan, Z., Ben-Hador, S., Goldberg, R. (1967). Rheumatoid arthritis in a Jerusalem population. Am.J. Epidemiol. 85:365.

Agranat, A.L., Bersohn, I., Lewis, S.M. (1957). Familial Disseminated Lupus Erythematosus. S.Afr.Med.J. 31:258.

Albright, F. (1947). Osteoporosis. Ann.Int.Med. 27:861.

Allander, E. (1970). A population survey of rheumatoid arthritis: epidemiological aspects of this syndrome, its pattern, and effect on gainful employment. Acta Rheum.Scand.Suppl. 15. 1970.

Allander, E., Bjelle, A. (1981). Developments in epidemiological studies on Rheumatoid arthritis. Scand.J.Rheumatol. 10:257.

Al-Rawi, Z.S., Alazzaei, A.J., Alajili, F.M., Alwakil, R. (1978). Rheumatoid arthritis in population samples in Iraq. Ann.Rheum. Dis. 37:73.

Alspaugh, M.A., Tan, E.M. (1976). Serum antibody in rheumatoid arthritis reactive with a cell associated antigen. Demonstration by precipitations and immunofluorescence. Arthritis Rheum. 19:711.

Alspaugh, M.A., Jensen, F.C., Rabin, H., Tan, E.M. (1978). Lymphocytes transformed by Ebstein-Barr virus: induction of nuclear antigen reactive with antibody in Rheumatoid arthritis. J.Exptl.Med. 147:1018.

Joint Motion Method of Measuring and Recording. American Academy of Orthopaedic Surgeons. 1965.

Anderson, I.F. (1971). Rheumatoid arthritis in the Bantu. S.Afr.Med.J. 44:1227.

Anderson, I.F., Klintworth, G.K. (1961). Hypovitaminosis A. in a family with tylosis and clinodactyly. Brit.Med.J. 1:1293.

Anderson, I.F., Anderson, R. (1981). A case of mixed connective tissue disease. S.Afr.Med.J. 60:806.

Anglo-American Corporation : Annual Medical Report 1981.

Anumonye Amechi. (1964). Juvenile Rheumatoid Arthritis in Nigerian children. West Afr.Med.J. 13:95.

Avioli, L.V. (1977). Osteoporosis: pathogenesis and therapy in Metabolic Bone Disease edited by Avioli, L.H., Krame, S.M. New York Academic Press 1977 p.307.

Bacon, F. (1658). Natural history and experimental of life and death or of the prolongation of life. W.Lee and Mosely. London.

Bagg, L.R., Hansen, D.P., Lewis, C., Houba, V. (1979). Rheumatoid arthritis in Kenya. Clinical observations. Ann.Rheum.Dis. 38:23.

Barland, P. (1975). The clinical significance of anti SM and anti RNP antibodies. (Abstr) Arth.Rheum. 18:384.

Barnett, E., Nordin, B.E.C. (1960). The radiological diagnosis of osteoporosis, a new approach. Clin.Radiol. 11:166.

Barnicot, N.A., Hardy, N.H., (1955). Position of the Hallux in West Africans. J.Anatomy 89:355.

Barry, M.E., Schneider, J. (1970). Meningococcal infection in African miners. Med. Proceedings 16:177.

Batchelor, J.R., Wooley, P., Panayi, G.S., Griffin, J., Laurent, M.R., (1979). HLA-DR alleles and their clinical significance in rheumatoid arthritis in Immunopathogenesis of rheumatoid arthritis edited by Panayi, G.S., and Johnson, P.M. Reedbooks p.19.

Bauer, G.C.H. (1960). Epidemiology of fracture in aged persons. Clin. Orthop. 17:219.

Beasley, R.P., Retailliau, H., Healey, L.A. (1973). Prevalence of rheumatoid arthritis in Alaskan Eskimos. Arthritis Rheum. 16:737-742.

Beasley, R.P., Wilkens, R.F., Bennett, P.H. (1973). High prevalence of rheumatoid arthritis in Yakima Indians. Arthritis Rheum. 16:743-748.

Beighton, P., Horan, F. (1970). Dominant inheritance in generalised articular hypermobility. J.Bone.Jnt.Surg. 52B: 145-147.

Beighton, P., Solomon, L., Soskolne, A. (1973). Articular mobility in an African population. Ann.Rheum.Dis. 32:413.

Beighton, P., Solomon, L., Soskolne, C.L., Sweet B. (1973). Serum uric acid concentration in a rural Tswana Community in South Africa. Ann. Rheum. Dis. 32:346.

Beighton, P., Sacks, S., (1979). Gauchers disease in Southern Africa. S.Afr.Med.J. 48:1295.

Beighton, P., Solomon, L., Soskolne, C.L., Sweet, B., Robin, G. (1974). Serum uric acid concentration in an urbanised South African Negro population. Ann.Rheum.Dis. 33:442.

Beighton, P., Soskolne, C.L., Solomon, L., Sweet, B. (1974). Serum Uric Acid in a Nama (Hottentot) Community in South West Africa. S.Afr.J.Sc. 70:281.

Beighton, P., Solomon, L., Valkenburg, H.A. (1975). Rheumatoid arthritis in a rural South African Negro population. Ann.Rheum. Dis. 34:126.

Beighton, P., Daynes, G., Soskolne, C.L. (1976). Serum uric acid concentrations in a Xhosa Community in the Transkei of South Africa. Ann.Rheum.Dis. 35:77.

Beighton, P., Durr, L., Hamersma, H. (1976). The clinical features of sclerosteosis. Ann.Int.Med. 84:393.

Benatar, S.R. (1977). Sarcoidosis in South Africa, a comparative study in Whites, Blacks and Coloureds. S.Afr.Med.J. 52:602.

Benatar, S.R. (1980). A comparative study of sarcoidosis in White, Black and Coloured South Africans in Eight International Conference in Sarcoidosis and other Granulomatous Diseases edited by W. Jones-Williams and B.H. Davies. Alpha Omega Publishing Ltd. 1980.

Ben-Dov, I., Berry, E. (1980). Acute rheumatic fever in adults over the age of 45 years: an analysis of 23 patients together with a review of the literature. Seminars Arthritis Rheum. 10:100.

Bennett, P.H., Burch, T.A., (1968). Osteoarthrosis in the Blackfeet and Pima Indians in Population Studies of the rheumatic diseases edited by Bennett, P.H., and Wood, P.H.N. Excerpta Medica Foundation Amsterdam p.407.

Bennett, G.A., Waine, H., Bauer, W. (1942). Changes in the knee joint at various ages, with particular reference to the nature and development of degenerative joint disease. Commonwealth Fund. New York.

Bennett, P.H., Wood, P.H.N. (1968). Population studies of the Rheumatic Diseases: Proceeding of the third international symposium. Excerpta Medica Foundation 1968 p.453.

Berman, L. (1975). Paper presented at the 5th Conference of the S.A. Rheumatism and Arthritis Ass. Cape Town. May 10-14th.

Bhettay, E., Thomson, A.J.G. (1972). Juvenile Rheumatoid arthritis in the Western Cape. Paper presented at the 3rd South African Rheumatism and Arthritis Conference July 19-22, Johannesburg.

Bird, H.A., Tribe, C.R., Bacon, P.A. (1978). Joint hypermobility leading to osteoarthrosis and chondrocalcinosis. Ann.Rheum.Dis. 37:203.

Bisno, A.L., Stollerman, G.H. (1975). Streptococcal antibodies in the diagnosis of rheumatic fever in Laboratory diagnostic proceedures in the Rheumatic Diseases edited by Cohen, A.S. p.20+ 2nd edition Little Brown Co. Boston.

Bland, J.H., Merritt, J.A., Boushey, D.R., (1977). The painful shoulder. Seminar Arthritis Rheum. 7:21.

Blumsohn, D. (1976). The pattern of joint disease in Black patients at Tembisa Hospital. Presented at the 5th Conference of the S.A. Rheumatism and Arthritis Ass. May 10-14th, Cape Town.

Bohan, A. (1981). The private practice of rheumatology: the first 1000 patients. Arthritis Rheum. 24:1304.

Bosworth, D.M. (1955). The role of the orbicular ligament in tennis elbow. J.Bone.Jnt.Surg: 37A. 527.

Bosworth D.M. (1965). Surgical treatment of tennis elbow, a follow-up study. J.Bone.Jnt.Surg: 47A. 1533.

Botha, M.C., Pritchard, J. (1972). Blood group gene frequencies : an indication of the genetic constitution of population samples in Cape Town. S.Afr.Med.J. 46. Suppl.1.

Bowes, W.G., Jabkovits, P. (1952). Osteitis tuberculosa multiplex cystoides. S.Afr.Med.J. 26.

Boyd, H.B., McCloed, A.C., (1973). Tennis Elbow. J.Bone.Jnt.Surg. 55A:1183.

Boyle, A.C. (1969). Disorders of the shoulder joint. Brit.Med.J. 3:283.

Bresnihan, B., Bunn, C., Snaith, M.L., Hughes, G.R.V. (1977). Anti-ribonucleoprotein antibodies in connective tissue diseases : estimation by counter immunoelectrophoresis. Br.Med.J. 1:610.

Bridgeman, J.F. (1972). Periarthritis of the shoulder in diabetes mellitus. Ann.Rheum.Dis. 31:69.

Briggs, B., Du Toit E., Meyers, O.L. (1980). Unpublished data.

Brighton, S.W., Louw, E.I. (1981). Social and rehabilitational aspects of rheumatoid arthritis. S.Afr.Med.J. 60:103.

Brooke, E.M. (1953). Incidence of rheumatic disease. Monthly Bull. Ministry of Health, London. 12:114.

Brozek, J., Keys, A., (1951). The evaluation of Leanness - Fatness in man : norms and interrelationships. Brit.J.Nutr. 5:194.

Buchanan, M.J., Isdale, I.C., Rose, B.S. (1965). Serum uric acid estimation : chemical and enzymatic methods compared. Ann.Rheum.Dis. 24:285.

Buchel, E. (1970). Multicentric Reticulo-histiocytosis (lipoid-dermato-arthritis) a clinical conundrum. S.Afr.Med.J. 44:1434.

Bulgen, D., Hazleman, B., Ward, M., McCallum, M. (1978). Immunological studies in the frozen shoulder. Ann.Rheum.Dis. 37:135.

Bunim, J.J., Burch, T.A., O'Brien, W.M. (1964). Influence of genetic and environmental factors on the occurrence of rheumatoid arthritis and rheumatoid factor in American Indians. Bull.Rheum.Dis. 15:349.

Burch, T.A., O'Brien, W.M., Need, R., Kurland, L.T. (1966). Hyperuricaemia and gout in the Manana Islanders. Ann.Rheum.Dis. 25:114.

Burnet, F.M. (1970). An immunological approach to ageing. Lancet 2:358.

Burnham, T.K. (1972). Anti-nuclear antibodies in patients with malignancies (Letter). Lancet 2:436.

Bywaters, E.G. (1973). Total management of the arthritic patient edited by Erlich, G.E. Lippincot Co. Philadelphia. Foreword.

Calin, A., Fries, J.F. (1975). The striking prevalence of ankylosing spondylitis in 'healthy' W27 positive males and females. A controlled study. N.Engl.J.Med. 293:835.

Calin, A., Porta, J., Fries, J.F., Schurman, D.J. (1977). Clinical history as a screening test for ankylosing spondylitis. J.Am.Med.Ass. 237:2613.

Cameron, T.R., Mazess, R.B., Sorenson, J.A. (1968). Precision and accuracy of bone mineral determination by direct photon absorphometry. Invest.Radiol. 3:141.

Cammarata, R.J., Rodnan, G.P., Fennell, R.H. (1964). Serological changes and serum protein concentrations in the aged. Arthritis Rheum. 7:297.

Canoso, J.J. (1981). Bursae, Tendons and ligaments in The Biology of the Joint. Edited by Hasselbachar, P., W.B. Sanders Co. 1981. pp.189.

Carr, C.R., Boyd, B.M. (1968). Correctional osteotomy for metarsus primus varus and hallux valgus. J.Bone.Jnt.Surg. 50A:1353.

Cassidy, M., Gregory, M.C., Harley, E.H. (1980). Primary over-production of urate caused by a partial defficiency of Hypox-anthine-guanine phosphoribosyl transferase. A.Afr.Med.J. 57:948.

Castle, W.M., Bernberg, H.C. (1969). Morbidity patterns among Europeans in Salisbury. Centr.Afr.J.Med. 15:8.

Caughey, D.E. (1974). The arthritis of Constantine IX. Ann. Rheum.Dis. 38:77.

Chalmers, I.M., McNeill, W.G. (1976). Rheumatoid arthritis in Africans: Recent experience at King Edward VIII Hospital. Presented at the 5th Conference of the S.A. Rheumatism and Arthritis Association May 10-14. Cape Town.

Chalmers, I.M., Pudifin, D.J., Shephard, E.G. (1977). Rheumatoid factor in non-rheumatoid Black patients. S.Afr.Med.J. 51:617.

Chalmers, I.M., Seedat, Y.K., Mudliar, M.Y. (1977). Ankylosing Spondylitis in three Zulu men negative for the HLA B27 antigen. S.Afr.Med.J. 52:567.

Chalmers, I.M., Pudifin, D.J., Shephard, E.G. (1978). The sheep cell agglutination test in non-rheumatoid Black patients. S.Afr.Med.J. 54:515.

Chalmers, I.M., Bhoola, K.D., Parsoo, I. (1979). Eosinophilic fasciitis. S.Afr.Med.J. 55:262.

Chalmers, I.M. (1980). Ankylosing spondylitis in Black Africans. Arthritis Rheum. 23:1366.

Chalmers, J., Ho, K.C. (1970). Geographic variations in senile osteoporosis : The association with physical activity. J.Bone.Jnt. Surg. 52B:667.

Chalmers, T.M. Danchot, J., Kellgren, J.H., King, D., Pikhlar, E., Sievers, K., Strevens, E., Tait, B., Wood, P.H.N. (1970). Test of diagnostic criteria - experience in England and Wales. Ann.Rheum.Dis. 29:200. Abstr.

Charcot, J.M. (1867). Lecons sur les maladies des Vieillards et les maladies chroniques. Delahaye. Paris.

Charcot, J.M. (1889). Maladies des Vieillards goutte et rheumatism, in Oeuvres complètes V7. Lescrosuier et Babè. Paris.

Chester, E., Levin, S., du Plessis, L., Freiman, I., Rogers, M., Joffe, N. (1966). The pattern of rheumatic heart disease in the urbanised Bantu of Johannesburg. S.Afr.Med.J. 40:899.

Cobb, S., Warren, J.E., Merchant, W.R., Thompson, D.J. (1957). An estimate of the prevalence of rheumatoid arthritis. J.Chron.Dis. 5:636.

Cobb, S. (1965). The epidemiology of rheumatoid arthritis. Arthritis Rheum. 8:76.

Codman, E.A. The Shoulder. Boston Todd. 1934.

Cohen, A.S., Reynolds, W.E., Franklin, E.C., Kulka, J.P., Ropes, M.W., Shulmari, L.E., Wallace, S.L. (1971). Preliminary criteria for the classification of systemic lupus erythematosus. Bull. on the Rheumatic Dis. 21:643.

Cohen, L.M., Mittal, K.K. Schmid, F.R., Rogers, L.E. Cohen, K.L. (1976). Increased risk for spondylitis stigmata in apparently healthy HLA W27 men. Ann.Int.Med. 84:1.

Cohn, S.H., Ellis, K.J., Wallach, S., Zanzi, I., Aitkins, H.L., Aloia, J.F. (1974). Absolute and relative deficit in total skeletal calcium and radial bone mineral content in osteoporosis. J.Nucl.Med. 15:428.

Cohn, S.H., Aloia, J.F., Letteri, J.M. (1978). Non-invasive measurements of bone mass and their clinical significance (Editorial). Calc.Tiss.Research 26:1.

Commerford, P.J., Meyers, O.L. (1977). Arthropathy associated with familial cold urticaria. S.Afr.Med.J. 51:105.

Consalazio, C.F., Johnson, R.E., Pecora, L.J. (1963). Body composition proceedures in Physiologic measurements of metabolic functions in man. McGraw Hill Book Co. New York 1163. p.300 et seq.

Cooke, T.D.V., Bennett, E.L., Ohno, O. (1980). Identification of immunoglobulins and complement components in articular collagen in tissues of patients with idiopathic osteoarthrosis in the aetiopathogenesis of osteoarthrosis. Edited by Nuki, G. p.144. Pitman Medical 1980.

Cooke, T.D.V. (1981). Immune deposits in osteoarthritic cartilage - their relationship to synovitis and disease site and pattern. Sem.Arthritis Rheum. 11 (Suppl.1.):109.

Couchman, K.G., Wigley, R.D., Prior I.A.M. (1970). Auto antibodies in the Carterton population survey. J.Chron.Dis. 23:45.

Crock, H.V. (1964). Post traumatic erosion of cartilage. J.Bone. Jnt.Surg. 46B:530.

Cromartie, W.J.,Craddock, J.G., Schwab, J.H., Anderle, S.K., Yans, C.H. (1977). Arthritis in rats after systemic injection of streptococcal cells or cell walls. J.Exp.Med. 146:1585.

Cyriax, J.H. (1936). The pathology and treatment of tennis elbow. J.Bone.Jnt.Surg. 18:921.

Cyriax, J. (1969). Textbook of orthopaedic medicine Vol.1. pages 219-291. 5th ed. 1969. Baillière, Tindall & Cassell.

Davidson, S., Passmore, R., Brock, J.F., Truswell, A.S. (1975). Human Nutrition and Dietetics. 6th Ed. Churchill Livingstone. Edinburgh, London, New York. 1975. p.564.

Davis, E. (1970). Criteria of rheumatic fever. Lancet 1:1043.

De Graff, F. (1959). Proceedings of the ISRA Symposium in the social aspects of chronic rheumatic joint affections. International Congress series No: 23. New York. Excerpta Medica Foundation p.7.

De Graff, R., Laine, V.A.I., Lawrence, J.S. (1963). Epidemiology of chronic rheumatism. Edited by Kellgren, J.H., Jeffrey, and Ball, J. p.228. Blackwell Scientific Publication. Oxford.

Dent, C.E. (1955). Idiopathic osteoporosis. Proc.Roy.Soc.Med. 48:574.

De Quecker, J. (1976). Quantative radiology : radio-grammetry of cortical bone. Brit.J.Radiol. 49:912.

De Palma, A. (1973). Surgery of the shoulder. 2nd Ed. Lippinott 1973.

Dick, B., Spencer, I.W.F., Watermeyer, G.S., Bourne, D.E., Wolff, E.M.P., Moyle, G.D. (1978). Chronic illness in non-institutionalised persons : Part 1. S.Afr.Med.J. 53:892.

Dieppe, P.A., Huskisson, E.C., Crocker, P.R., Willoughby, D.A. (1976). Apatite deposition disease : A new arthropathy. Lancet 1:266.

Donaldson, I.A., Nassim, J.K. (1954). The artificial menopause with particular reference to spinal osteoporosis. Brit.Med.J. 1:1228.

Drew, R.A. (1972). Erysipelothrix arthritis in pigs as a comparative model for rheumatoid arthritis. Proc.Roy.Soc.Med. 65:994.

Dunstan, H.P., Taylor, R.D., Corcoran, A.C., Page, I.H. (1954). Rheumatic and febrile syndrome during prolonged Hydrallazine treatment. J.Am.Med.Ass. 154:23.

Du Toit, E. (1981). Personal communication.

Du Toit, G.T. (1979). Hip disease of Mseleni. Clin.Orthop. 141:223.

Du Toit, S.N. (1973). A one year survey of a rural orthopaedic Clinic in Zululand. S.Afr.Med.J. 47:2189.

Eales, L. (1951). Brucella mellitensis infection presenting as an acute arthritis of the hip joint. S.Afr.Med.J. 25:143.

Elliott, G.A. (1942). Tick-bite fever. Clinical descriptions of an outbreak of 296 cases. Clin.Proceedings 1:70.

Elves, M.W., Bucknill, T., Sullivan, M.F. (1975). In vitro inhibition of leucocyte migration in patients with inter-vertebral disc lesions. Orthop.Clin.North Amer. 6:59.

Engel, A. (1968). Rheumatoid arthritis in U.S. adults 1960-1962 in Population studies of the rheumatic diseases. Edited by Bennett, P.H. and Wood, P.H.N. Excerpta Medica Foundation, Amsterdam. pp.83.

Epstein, W.V., Henke, C.J. (1981). The nature of U.S. rheumatology practice 1977. Arthritis Rheum. 24:1177.

Erasmus, L.D. (1957). Scleroderma in gold miners on the Witwatersrand with particular reference to pulmonary manifestation. S.Afr. J. Lab.Clin.Med. 3:209.

Erlich, G.E. (1972). Inflammatory osteoarthritis. 1. The Clinical Syndrome. J.Chron.Dis. 25:317.

Evans, R.A., McDonnell, G.D., Schieb, M. (1978). Metacarpal cortical area as an index of bone mass. Brit.J.Radiol. 51:428.

Exton-Smith, A.N., Millard, P.H., Payne, P.R., Wheeler, E.F., (1969). Method of measuring quantity of bone and pattern of development and loss of bone with age. Lancet 2:1153.

Fabius, A.J.M., Gaulhofer, W.K. (1971). Systemic lupus erythematosus induced by psychotropic drugs. Act Rheum.Scand. 17:137.

Falconer, A.W., Ryrie, B.J. (1937). A report on a familial type of generalised osteo-sclerosis. Medical Press and Circular 195:12.

Fellingham, S.A., Elphinstone, C.D., Wittman, W. (1973). Mseleni Joint Disease : background and prevalence. S.Afr.Med.J. 47:2173.

Fernandez-Madrid, F., Mattioli, M. (1976). Anti-nuclear antibodies (ANA) : Immunologic and clinical significance. Sem.Arthritis Rheum. 6:83.

Fessel, W.J., Barr, G.D. (1977). Uric Acid Lean Body weight and creatinine interactions results from regression analysis of 78 variables. Semin.Arthr.Rheum. 7:115.

Fincham, J.E., Van Rensburg, S.J., Marasas, W.F.O. (1981). Mseleni joint disease - a manganese deficiency? S.Afr.Med.J. 60:445.

Findlay, G.H., Oosthuisen W.J. (1951). Pachydermo peri ostitis. S.Afr.Med.J. 25:747.

Findlay, G.H., Price, E.A., Van Rensburg, C.R.J. (1951). Dermatomyositis with vesicular and bullous lesions. S.Afr.Med.J. 25:60.

First, M.R. (1973). Familial Lupus erythematosus. A.Afr.Med.J. 47:742.

Fisherman, E.W. (1960). Does the allergic diathesis influence malignancy? J.Allergy. 31:74.

Forbes, J. (1960). Ankylosing spondylitis : a case report. Centr.Afr.J.Med. 6:461.

Forman, M.B., Kalk, W.J. (1981). Yersinia arthritis mimicing acute rheumatic fever : a case report. S.Afr.Med.J. 59:576.

Forman, M.B., Lewin, J.R., Gear, A.J., Solomon L. (1981). Eosinophilic fasciitis in South Africa. S.Afr.Med.J. 59:540.

Fourie, E.D., Morrison, J.G.L. (1979). Rheumatoid arthritic syndrome after Chikungunya fever. S.Afr.Med.J. 56:130.

Friou, G.J. (1957). Clinical application of lupus serum-nucleoprotein reaction using fluorescent antibody technique. J.Clin. Invest. 36:890.

Friou, G.J. (1967). The LE cell factor and antinuclear antibodies in Laboratory diagnostic proceedures in the rheumatic diseases. Edited by Cohen, A.S. Little Brown Co. Boston. p.123.

Gaffney, J.C. (1978). Profile of Medical Practice. American Medical Association, Centre for Health Services Research and Development 1979.

Garden, R.S. (1961). Tennis elbow. J.Bone Jnt.Surg. 43B:100.

Gardner, R.C. (1970). Confirmed case and diagnosis of pseudo carpal tunnel (sublimis) syndrome. N.Engl.J.Med. 282:858.

Garn, S.M., Rohmann, C.G., Wagner, B., Ascoli, W. (1967). Continuing bone growth throughout life : a general phenomenon. Amer.J.Physical anthropol. 26:313.

Garrod, A.B. (1859). The nature and treatment of gout and rheumatic gout. p.532, Walton and Maberly, London.

Gelfand, M. (1962). Three cases of rarefaction of the skeleton in Africans : the association with severe urinary bilharziasis. Centr.Afr.J.Med. 8:9.

Gelfand, M. (1963). Acute non-specific arthritis in the African. Centr.Afr.J.Med. 9:276.

Gelfand, M. (1969). Medical arthritis in an African practice. Centr.Afr.J.Med. 15:131.

Gelfand, M. (1970). Commenting on a case of scleroderma presented at Harari Staff Round. Centr.Afr.J.Med. 16:241.

Gertzbein, S. (1981). The antigeniality of the intervertebral disc. Sem.Arthritis Rheum. 11 (Suppl. 1):111.

Gilbertson, E.M.M. (1975). Development of periarticular osteophytes in experimentally induced osteoarthritis in the dog : a study using microradiographic, microangiopathic and fluorescent bone labelling techniques. Ann.Rheum.Dis. 34:12.

Glick, E.N. (1967). Asymmetrical rheumatoid arthritis after poliomyelitis. Brit.Med.J. 3:26.

Glynn, L.E. (1968). The chronicity of inflammation and its significance in rheumatoid arthritis. Ann.Rheum.Dis. 27:105.

Goetz, R.H. (1945). The pathology of progressive systemic sclerosis (generalised scleroderma). Clin.Proceedings 4:337.

Gofton, J.P., Robinson, H.S., Price, G.E., (1964). A study of rheumatic disease in a Canadian Indian Population II. Ann.Rheum.Dis. 23:364.

Goldblatt, J. (1980). Gauchers Disease in the AshKenazi Jewish Community of South Africa. MD. Thesis. University of Cape Town.

Goldie, Ian. (1964). Epicondylitis lateralis humeri. Acta.Chir. Scand.Suppl. 339:7.

Goodall, J.W.D. (1956). Joint swellings in Africans. Centr.Afr.J. Med. 2:220.

Gordon, T. (1968). Osteoarthrosis in U.S. adults in Population studies of the Rheumatic Diseases. Ed. by Bennett, P.H., and Wood, P.H.N. Excerpta Medica Foundation, Amsterdam. p.391.

Gordon-Smith, P. (1964). Ongewone verskynsel by sarkoiedose. Geneeskunde 6:346.

Gottschalk, F.A.B., Sallis, J.G., Beighton, P.H., Solomon, L. (1980). A comparison of the prevalence of hallux valgus in three South African populations. S.Afr.Med.J. 57:355.

Grahame, R., and Scott, J.T. (1970). Clinical survey of 354 patients with gout. Ann.Rheum.Dis. 29:461.

Grahame, R., Edwards, J.C., Pitcher, D., Gabell, A., Harvey, W., (1981). A clinical and echocardiographic study of patients with the hypermobility syndrome. Ann.Rheum.Dis. 40:54.

Green, L., Meyers, O.L., Gordon, W., Briggs, B., (1981). Arthritis in psoriasis. Ann.Rheum.Dis. 40:366.

Greenwood, B.M. (1969). Acute tropical polyarthritis. Quart.J.Med. 38:295.

Greenwood, B.M. (1969). Polyarthritis in Western Nigeria : 1. Rheumatoid Arthritis. Ann.Rheum.Dis. 28:488.

Grusin, H., Kincaird-Smith, P.S. (1954). Scurvy in Adult Africans. Am.J.Clin.Nutr. 2:323.

Grusin, H., Samuel, E. (1957). A syndrome of osteoporosis in Africans and its relationship to scurvy. Am.J.Clin.Nutr. 5:644.

Hadler, N.M. (1976). A pathogenetic model for erosive synovitis; lessons from animal arthritides. Arthritis Rheum. 19:256.

Hahn, T.J., Hahn, B.H. (1976). Osteopenia in patients with rheumatic diseases : principles of diagnosis and therapy. Sem.Arthritis.Rheum.6:165.

Hall, A.P., Barry, P.E., Dawber, T.R., McNamera, P.M. (1967). Epidemiology of gout and hyperuricaemia a long term Population study. Am.J.Med. 42:27.

Hall, L. (1966). Polyarthritis in Kenya. E.Afr.Med.J. 43:161.

Hamilton-Fairley, G. (1972). Auto-antibodies in malignant disease. Brit.J.Haematol. 23 (suppl):231.

Hammond, M.G., Appadoo, D., Brain, P. (1972). HLA Antigens and antibodies in South African Bantu. Transplantation 14:159.

Harari Staff Round (1971). Tropical pyomyositis. Centr.Afr.J. Med. 17:136.

Hardy, R.H., Clapham, J.C.R. (1951). Observations on Hallux Valgus. J.Bone Jnt.Surg. 33B:272.

Harris, E.D. (1981). Pathogenesis of rheumatoid arthritis in Textbook of Rheumatology Vol.1. Edited by Kelley, W.N., Harris, E.D., Ruddy, S., and Sledge, C.B. W.B. Saunders Co. Philidelphia. p.896.

Harris, W.H., Heaney, R.P. (1969). Skeletal renewal and metabolic bone disease. N.Engl.J.Med. 280:193.

Harrison, M.H.M., Schajowicz, F., Trueta, J. (1953). Osteoarthritis of the hip : a study of the nature and the evolution of the disease. J.Bone.Jnt.Surg. 35B:598.

Harvey, J., Lotze, M., Stevens, M.B. (1981). Rheumatoid arthritis in a Chippewa Band. Arthritis.Rheum. 24:717.

Hayes, M.M.M., Gwata, T., Gelfand, M. (1978). Takayashu's disease in association with probable Stills syndrome in a nine year old African male. Centr.Afr.J.Med. 24:144.

Haygarth, J. (1805). A clinical history of disease. Part 1st being 1. A clinical history of acute rheumatism. 2. A clinical history of the nodosity of joints. Cadell Davies. London.

Hazleman, B. (1972). The painful stiff shoulder. Rheumatol & Physical Med. 11:413.

Hebert, W.J. (1973). Passive haemogglutination with special reference to the tanned cell technique in Handbook of experimental Immunology. Ed. by Weir, D.M. 2nd edition. page 201. Blackwell Scientific Publications, London.

Hegner, R.A. (1924). Flexed fingers. J.Heredit. 15:481.

Hellgren, L. (1970). Prevalence of rheumatoid arthritis in densely and thinly populated areas in Sweden. Act.Rheum.Scand. 16:18.

Henrard, J.C., Bennett, P.H., Burch, T.A. (1975). Rheumatoid arthritis in Pima Indians of Arizona. An assessment of the clinical components of the New York Criteria. Int.J.Epid. 4:119.

Hift, W., Watson, K.C. (1968). Systemic Lupus Erythematosus in a family. S.Afr.Med.J. 42:826.

Holborow, J., Johnson, G.D., Farrow, L.J. (1971). Immunofluorescent detection of auto-antibodies in the sera of acute infective hepatitis patients. Ann.NY.Acad.Sci. 177:214.

Holborow, J. (1972). Smooth muscle auto antibodies viral infections and malignant disease. Proc.Roy.Soc.Med. 65:481.

Hollister, L.E., Engelman, E.P. (1958). Rheumatoid spondylitis without RA of peripheral joints : relationship to rheumatic fever. J.Chron.Dis. 8:334.

Hooper, B., Whittingham, S., Mathews, J.D., MacKay, I.R., Curnow, D.H. (1972). Autoimmunity in a rural community. Clin.Exptl.Immunol. 12:79.

Horan, F., Beighton, P. (1971). Recessive inheritance of generalised joint hypermobility. Rheumatol.Rehab. 12:47.

Horsfall, P.A.L. (1965). Dermatomyositis in the South African Bantu. S.Afr.Med.J. 39:695.

Hough, A.J., Banfield, W.G., Mottram, F.C., Sokoloff, L. (1974). The osteochondral junction of mammalian joints : an ultrastructural and microanalytical study. Lab.Investig. 31:685.

Hueston, J.T. (1963). Dupuytren's Contracture. Livingstone, Edinburgh & London 1963.

Hurwitz, M.D., Catchpole, M.V., Plit, M. (1982). The gonococcal arthritis - dermatitis syndrome : a case report. S.Afr.Med.J. 61:555.

Hyer, F.H., Gottlieb, N.L. (1978). Rheumatic disorders associated with viral infection. Sem.Arthritis Rheum. 8:17.

Isaacs, H., Heffron, J.J.A., Berman, L., Badenhorst, M., Pickering, A. (1975). Xanthine, hypoxanthine and muscle pain : histochemical and biochemical observations. S.Afr.Med.J. 49:1035.

Iskrant, A.P., Smith, R.W. (Jr.) (1969). Osteoporosis in women 45 years and over related to subsequent fractures. Public Health Rep. 84:33.

Jacobs, A., Entwistle, C.C., Campbell, H., Waters, W.E., (1969). A random sample from Wales IV. Circulating gastric and thyroid and antinuclear factor. Brit.J.Haematol. 17:589.

Jenkins, T. (1972). Genetic polymorphisms of man in Southern Africa. MD.Thesis. University of London.

Jessop, S., Gordon, W. (1975). Multicentric reticulo-histiocytosis. Case report. S.Afr.Med.J. 49:2191.

Jessop, S., Meyers, O.L. (1973). Systemic Lupus erythematosus in Cape Town. S.Afr.Med.J. 47:222.

Johnston, I. (1956). Further studies of the inheritance of hand and foot anomalies. Clin.Orthop. 8:146.

Jones, B.S. (1971). Adolescent chondrolysis of the Hip Joint. S.Afr.Med.J. 45:196.

Jones, J.P., Jameson, R.M., Engelman, E.P. (1968). Alcoholism, fat embolism and avascular necrosis (Abstr.). J.Bone Jnt.Surg. 50A:1065.

Joyce-Clarke, N. (1975). Gonococcal arthritis. Paper presented at the 5th South African Rheumatism and Arthritis Congress, Cape Town. May 10-14th.

Kahlmeter, G. (1932). Du röle Jouée ensuède par le rheumatisme articulaire chronique comme cause d'incapacitè permanente de travail dans les diverses groupes professionels. Acta.Rheum.Scand. 4:34.

Kamiyama, S., Kobayashi, S., Takahashi, E. (1968). Osteoporosis in hypertensive and nonnhypertensive subjects : an epidemiological approach to the aetiology of osteoporosis. Tohoku J.Exp.Med. 94:225.

Kanyerezi, B.R. (1969). Rheumatoid arthritis in Uganda. E.Afr. Med.J. 46:71.

Kaplan, E.L., Top, F.H., Dudding, B.A., Wannamaker, L.W. (1971). Diagnosis of streptococcal pharyngitis : differentiation of active infection from the carrier state in the symptomatic child. J.Inf.Dis. 123:490.

Kaplan, J.M., Wachtel, H.L., Czarnecki, S.W., Sampson, J.J. (1965). Lupus-like illness precipitated by procainamide hydrochloride. J.Am.Med.Ass. 191:444.

Keen, P. (1944). Osteoarthritis in the Bantu. S.Afr.Med.J. 18:267.

Keeton, G.R. (1979). Diagnostic and therapeutic problems of polyarteritis nodosa. S.Afr.Med.J. 56:634.

Kellgren, J.H., Lawrence, J.S. (1952). Rheumatism in Miners: II X-ray study. Brit.J.Industr.Med. 9:197.

Kellgren, J.H., Moore, R. (1952). Generalised osteo-arthritis and Heberden's nodes. Brit.Med.J. 1:181.

Kellgren, J.H., Lawrence, J.S., Aitken-Swan, J. (1953). Rheumatic complaints in an urban population. Ann.Rheum.Dis. 12:5.

Kellgren, J.H., Bier, F. (1956). Radiological signs of rheumatoid arthritis : a study of observer differences in the reading of hand films. Ann.Rheum.Dis. 15:55.

Kellgren, J.H., Lawrence, J.S. (1957). Radiological assessment of osteoarthritis. Ann.Rheum.Dis. 16:494.

Kellgren, J.H., Jeffrey, M.R., Ball, J. (1963). The epidemiology of chronic rheumatism Vol.1. Blackwell, Oxford.

Kellgren, J.H., Jeffrey, M.R., Ball, J. (1963a). The epidemiology of chronic rheumatism Vol.2. Atlas of Standard radiographs of arthritis. Oxford. Blackwell

Kellgren, J.H. (1966). Epidemiology of rheumatoid arthritis. Arthritis Rheum. 9:658.

Kellog-Speed, (1929). Brit.Med.J. 2:1122.

Kemble, F. (1968). Electrodiagnosis of the carpal tunnel syndrome. J.Neurol.Neurosurg.Psychiat. 31:23.

Kirk, J.A., Ansell, B.M., Bywaters, E.G.L. (1967). The hypermobility syndrome : musculo skeletal complaints associated with generalised joint hypermobility. Ann.Rheum.Dis. 26:419.

Klemp, P., Meyers, O.L. (1976). Ankylosing spondylitis in a Xhosa father and daughter. S.Afr.Med.J. 50:1439.

Kozin, F., McCarthy, D.J., Sims, J., Gennant, H. (1976). The reflex sympathetic dystrophy syndrome 1. Clinical and histological studies. Am.J.Med. 60:321.

Labella, F.S., Lindsay, W.G. (1963). The structure of human aortic elastin as influenced by age. J.Gerontol. 18:111.

Lachman, E. (1955). Osteoporosis : the potentialities and limitations of its roentgenologic diagnosis. Am.J.Roentgenol. Radium Ther-Nucl.Med. 74:712.

Lachman, P.J., Hobart, M.J., Aston, W.P. (1973). Complement technology in Handbook of experimental Immunology edited by Weir, D.M. page 59. Blackwell Scientific Publications, London.

Laine, V. (1968). Report from the subcommittee on diagnostic criteria for osteoarthrosis in Population studies of the Rheumatic diseases edited by Bennett, P.H., Wood, P.H.N. Excerpta Medica Foundation. Amsterdam. 1968. p.417.

Lambert, H.P. (1968). Syndrome with joint manifestation in mycoplasma pneumonial infection. Brit.Med.J. 3:156.

Lategan, L.R. (1971). Nutritional observations made on Bantu recruits on engagement with special reference to osteoporosis. Proc.Mine Med.Officers Ass. S.A. 51:105.

Lawrence, J.S. (1955). Rheumatism in Coal Miners III Occupational factors. Brit.J.Industr.Med. 12:249.

Lawrence, J.S. (1961). Prevalence of rheumatoid arthritis. Ann.Rheum.Dis. 16:11.

Lawrence, J.S., Bremner, J.A., Ball, J., Burch, J.A. (1966). Rheumatoid arthritis in a subtropical population. Ann.Rheum.Dis. 25:59.

Lawrence, J.S. (1969)a. The epidemiology and genetics of rheumatoid arthritis in Rheumatology, Basel 2.1. Karger Basel/New York.

Lawrence, J.S. (1969).b. Disc degeneration : its frequency and relationship to symptoms. Ann.Rheum.Dis. 28:121.

Lawrence, J.S. (1977).c. Rheumatism in Populations. William Heineman. London 1977. p.462.

Lawrence, J.S. (1977).d. Rheumatism in Populations. William Heineman. 1977. p.462.

Lawrence, J.S. (1977).e. Rheumatism in Populations. William Heineman. London 1977. p.481.

Lawrence, J.S. (1977).f. Rheumatism in Populations. William Heineman. p.114.

Lawrence, J.S. (1977).g. Rheumatism in Populations. William Heineman. London. p.99.

Lawrence, J.S. (1977).h. Rheumatism in Populations. William Heineman. London p.206 and 215.

Lawrence, J.S. (1977).i. Rheumatism in Populations. William Heineman. London. p.298.

Lawrence, J.S. (1977).j. Benign polyarthritis in Rheumatism in populations J.S. Lawrence, Williams Heineman, London. p.272.

Lawrence, J.S., Sebo, M. (1980). The geography of osteoarthrosis in the Aetiopathogenesis of osteoarthrosis edited by Nuki, G. Pitman Medical p.155.

Leivisalo, M., Laitinen, O. (1975). Rheumatic fever in adult patients. Ann.Clin.Res. 7:244.

Lequesne, M., Dang, N., Bensasson, M., Mery, C. (1977). Increased association of diabetes mellitus with capsulitis of the shoulder and shoulder-hand syndrome. Scand.J.Rheumatol. 6:53.

Levy, L. (1966). Lumbar intervertebral disc in Africans. J.Neurosurgery 26:31.

Lindberg, B.J. (1970). Glycosaminoglycans of normal and frozen shoulder-joint capsule. Clin.Orthoped. 69:279.

Lockitch, G., Fellingham, S.A., Elphinstone, C.D., (1973). Mseleni joint disease : a radiological study of two affected families. S.Afr.Med.J. 47:2366.

Lockitch, G., Fellingham, S.A., Wittman, W., De Villiers, P.D., De Wet, I.S., Du Toit, G.T. (1973). Mseleni Joint disease : The pilot clinical survey. S.Afr.Med.J. 47:2283.

Lowenthal, M.N., Diamond, I.D. (1977). Gout and Hyperuricaemia in Blacks. S.Afr.Med.J. 52:832.

Lumholt, G. (1963). Psoriasis, spontaneous course and genetics. Copenhagen. G.E.C. Gad. 1963.

Lawrence, J.S., Behrend, T., Bennett, P.H., Bremner, J.M., Burch, T.A., Gofton, J.P., O'Brien, W.E., Robinson, H. (1966). Geographical studies in rheumatoid arthritis. Ann.Rheum.Dis. 25: 425.

Lawrence, J.S., Bremner, J.M., Ball, J., Burch, T.A., (1966). Rheumatoid arthritis in a subtropical population. Ann.Rheum.Dis. 25:59.

MacKay, W.D. (1966). The incidence of allergic disorders and cancer. Brit.J.Cancer. 20:434.

McCain, G.A., Bell, D.A., Chodirker, W.B., Komar, R.R. (1978). Antibody to extractable nuclear antigen in the rheumatic diseases. J.Rheumatol. 5:399.

McCarty, D.J., Gatter, R.A. (1966). Recurrent acute inflammation associated with focal apatite crystal deposition. Arthritis Rheum. 9:804.

McCarty, D.J. (1975). Diagnostic mimicry in arthritis - patterns of joint involvement associated with calcium phosphate dihydrate crystal deposits. Bull.Rheumatic Dis. 25:804.

McIntosh, B.M., Serafini, E.T., Dickinson, D.B., Weinbren, M.P. (1962). Antibodies against certain arbor viruses in sera from human beings resident in the coastal areas of Southern Natal and Eastern Cape Provinces of South Africa. S.Afr.J.Med.Sci. 27:77.

MacMahon, B., Pugh, T.F. (1970). Epidemiology : Principles and methods. Little Brown Company, Boston.

MacNab, I. (1973). Rotator cuff tendonitis. Ann.Roy.Coll.Surg. of Engl. 53:271.

Mankin, H.J. (1974). The reaction of articular cartilage to injury and to osteoarthritis. Part I. N.Engl.J.Med. 291:1285. Part 2. N.Engl.J.Med. 291:1335.

Mankin, H.S., Thrasher, A.Z. (1975). Water content and binding in normal and osteoarthritic human cartilage. J.Bone Jnt.Surg. 57A:76.

Marmion, B.P. (1976). A microbiologist's view of investigative rheumatology in Infection and Immunology in the rheumatic diseases. Edited by Dumonde, D.C. Oxford. Blackwell Scientific Publications, page 245.

Marshall, J.L., Olsson, S.E. (1971). Instability of the knee : a long term experimental study in dogs. J.Bone Jnt.Surg. 53A:1561.

Meachim, G. (1980). Ways of cartilage breakdown in human and experimental osteoarthrosis in the Aetiopathogenisis of osteo-arthrosis edited by G. Nuki. Pitman Medical 1980.

Meema, H.E., Meema, S. (1969). Cortical bone mineral density versus cortical thickness in the diagnosis of osteoporosis : a roentgenologic-desitometric study. J.Amer.Geriatric Soc. 17:120.

Meyers, O.L. (1980) Unpublished data.

Meyers, O.L., Chalmers, I. (1977). Jaccoud's Arthropathy. S.Afr. Med.J. 51:753.

Meyers, O.L. Commerford, P.J. (1977). Musculo-skeletal manifestations of bacterial endocarditis. Ann.Rheum.Dis. 36:517.

Meyers, O.L., Daynes, G., Beighton, P. (1977). Rheumatoid arthritis in a tribal Xhosa population in the Transkei, Southern Africa. Ann.Rheum.Dis. 36:62.

Meyers, O.L., Quantock, O.P. (1974). Chronic Scleroedema. S.Afr.Med.J. 48:164.

Mikkelsen, W.M., Dodge, H.J., Duff, I.F., Epstein, F.H., Napier, J.A. (1962). Abstr. Clinical and Serological estimates of RA in Tecumseh. Arthritis Rheum. 5:117.

Mikkelsen, W.M., Dodge, H.J., Valkenburg, H., Hines, S. (1965). Distribution of serum uric acid value in a population. Am.J.Med. 39:242.

Mikkelsen, W.M., Dodge, H.J., Duff, I.F., Kato, H. (1967). Estimates of the prevalence of rheumatoid diseases in the population of Tecumseh Michigan 1959n60. J.Chronic.Dis. 20:351.

Mikkelsen, W.M., Duff, I.F., Dodge, H.J. (1970). Age - specific prevalence of radiographic abnormalities of the joints of the hands, wrists and cervical spine of adult residents of Tecumseh. J.Chron.Dis. 23:151.

Mitchell, C.L., Fleming, J.L., Allen, R., Glenny, C., Sandford, G.A. (1958). Osteotomy - bunionectomy for hallux valgus. J.Bone Jnt. Surg. 40.A:41.

Moore, C.P., Wilkens, R.F. (1977). The subcutaneous nodule : its significance in the diagnosis of rheumatic disease. Sem.Arthritis Rheum. 7:63.

Morrison, J.G.L. (1974). Sarcoidosis in the Bantu. Necrotising and mutilating forms of the disease. Brit.J.Dermatol. 90:649.

Moseley, H.F. (1972). Shoulder Lesions. 3rd Rev. Ed. Edinburgh, Churchill Livingstone 1969, reprinted 1972.

Moskowitz, R.W., Davis, W., Sammarco, J., Martens, M., Baker, J., Mayor, M., Burstein, A.N., Frankel, V.N. (1973). Experimentally induced degenerative joint lesions following partial menisectomy in the rabbit. Arthritis Rheum. 16:397.

Muir, H. (1977). Molecular approach to the understanding of osteo-arthrosis. Ann.Rheum.Dis. 36:199.

Muller, A.S. (1970). Population studies on the prevalence of rheumatic diseases in Liberia & Nigeria. MD.Thesis. University of Leiden.

Murray-Leslie, C., Magaro, M., Wright, V. (1976). Progressive destruction of the femoral head in association with familial hypercholesterolaemia. Rheumatology and Rehabilitation 15:277.

Naidoo, P.D. (1981). Stills disease in an adult - a case report. S.Afr.Med.J. 60:551.

Naidoo, P.M., Chanyan, C. (1975). Typhoid polymyositis. S.Afr. Med.J. 49:1975.

Neviaser, J.S. (1945). Adhesive capsulitis of the shoulder : a study of the pathologicol findings in periarthritis of the shoulder. J.Bone.Jnt.Surg. 27A:211-222.

Nicholls, A., Scott, J.T. (1972). Effect of weight loss in plasma and urinary levels of uric acid. Lancet 2:1223.

Nicholls, A., Snaith, M.L. Yablonsky, H., Scott, J.T., (1973). Effect of stilboestrol on levels of uric acid in plasma and urine. Heberden Soc.Meeting. Nov. 1972.

Nordin, B.E.C. (1960). Bone as a tissue. Edited by K. Rodahl., J.T. Nicholson, E.M. Brown Jr. New York. McGraw-Hill. p.46.

Nordin, B.E.C., MacGregor, J., Smith, D.A., (1966). The incidence of osteoporosis in normal women in relation to age and the menopause. Quart.J.Med. (NS). 35:25.

Nordin, B.E.C. (1971). Clinical significance and pathogenesis of osteoporosis. Brit.Med.J. 1:571.

Nuki, G., Brookes, R., Buchanan, W.W. (1972). The economics of arthritis. Bull.Rheum.Dis. 23:726.

Nurse, G.T., Jenkins, T. (1974). Mseleni joint disease; population genetic studies. S.Afr.J.Sci. 70:360.

O'Brien, W.M., Bennett, P.H., Burch, T.A., Bunim, J.J. (1967). A genetic study of rheumatoid arthritis and rheumatoid factor in Blackfeet and Pima Indians. Arthritis Rheum. 10:163.

O'Brien, W.M., Clemett, A.R., Acheson, R.M. (1968). Symptoms and pattern of osteoarthrosis in the hand in the New Haven survey of joint disease in Population studies of the Rheumatic Diseases. Edited by Bennett, P.H. and Wood, P.H.N. Excerpta Medica Foundation, Amsterdam. p.398.

Olhagen, B., Månnson, I. (1968). Intestinal clostridium perfringens in rheumatoid arthritis and other collagen diseases. Acta Med.Scand. 184:395.

O'Sullivan, J.B., Cathcart, E.S. (1972). Prevalence of rheumatoid arthritis: followup evaluation of the effect of criteria on rates in Sudbury, Massachusetts. Ann.Int.Med. 76:573.

O'Sullivan, J.B., Francis, J.O'S., Kantor, N. (1965). Comparison of a colorimetric (automated) with an enzymic (normal) uric acid proceedure. Clin.Chem. 11:427.

Panayi, G.S., Wooley, P., Batchelor, J.R. (1978). Genetic basis of rheumatoid disease: HLA antigens, disease manifestations and toxic reactions to drugs. Brit.Med.J. 2:1326.

Parish, L.C. (1963). An historical approach to the nomenclature of rheumatoid arthritis. Based on data from a thesis of Landré-Beavais 1800. University of Paris. Arthritis Rheum. 6:138.

Pearson, C.M. (1956). Development of arthritis, peri-arthritis and periostitis in rats given adjuvants. Proc.Soc.Exp.Biol.Med. 91:95.

Percy-Lancaster, P.C. (1974). Arthritis Survey in Transkei and Ciskei. S.Afr.Med.J. 48:2355.

Phalen, G.S. (1966). The carpal tunnel syndrome : seventeen years experience in diagnosis and treatment of 654 hands. J.Bone.Jnt.Surg. 48A:211.

Phillips, P.E. (1976). Virus infections and rheumatic disease : possible models for the pathogenesis of rheumatoid arthritis in Modern Topics in Rheumatology. Edited by Hughes, G.R.V., William Heineman Medical Books, London p.8.

Phillips, H.B., Owen-Jones, S., Chandler, B. (1978). Quantitative histology of Bone : A computerised method of measuring the total mineral content of bone. Calcif.Tiss.Research. 26:85.

Piggot, H. (1960). The natural history of hallux valgus in adolescents and early adult life. J.Bone.Jnt.Surg. 42B:749.

Pijper, A. (1934). Tick-bite fever : a clinical lecture. S.Afr.Med.J. 13:551.

Pimstone, B.L. (1966). Systemic Lupus erythematosus exacerbated by oral contraceptives. S.Afr.J.Obstet.Gynaecol. 4:62.

Post, M. (1978). The shoulder : surgical and non-surgical management. Lea Febeiger 1978.

Prior, I.A.M., Rose, B.S., Davidson, F. (1964). Metabolic maladies in New Zealand Maoris. Brit.Med.J. 1:1065.

Prior, I.A.M., Rose, B.S., Harvey, H.P.B., Davidson, F. (1966). Hyperuricaemia, gout and diabetes abnormality in Polynesian people. Lancet 1:333.

Radin, E.L., Parker, H.G., Paul, I.L. (1971). Pattern of degenerative arthritis. Preferential envolvement of distal finger joints. Lancet 1:377.

Radin, E.L., Parker, H.G., Pugh, J.W., Steinberg, R.S., Paul, I.L., Rose, R.M., (1973). Articular cartilage more easily damaged by impact than abrasion. J.Biomechanics 6:51.

Ram, J.S., (1967). Ageing and immunological phenomena; a review. J.Gerontol. 22:92.

Rathburn, J.B., MacNab, I. (1970). The microvascular pattern of the rotator cuff. J.Bone.Jnt.Surg. 52B:540.

Ridge, M.D., Wright, V. (1966). The ageing of skin : a bio-engineering approach. Gerontologia 12:174.

Riley, M. (1976). Acute non-specific polyarthritis. Centr.Afr. J.Med. 22:1.

Ritchkin, J. (1956). Rubella rheumatism. Centr.Afr.J.Med. 2:85.

Roberts, C.J. (1979). Epidemiology for Clinicians. Pitman Medical.

Robertson, M., Thomas, A. (1976). Osteitis deformans in the South African Negro. Orthopaedia 4:1.

Rogers, J., Watt, I., Dieppe, P. (1981). Arthritis in Saxon and mediaeval skeletons. Brit.Med.J. 283:1668.

Roles, N.C., Maudsley, R.H. (1973). Radial Tunnel Syndrome. J.Bone Jnt.Surg. 54B:499.

Romer, F. (1922). Observations on Tennis elbow. Lancet 2:67.

Ropes, M. (1959). Diagnostic criteria for rheumatoid arthritis. 1958 Revision. Ann.Rheum.Dis. 18:49.

Rose, B.S., Prior I.A.M. (1963). A survey of rheumatism in rural New Zealand Maori Community. Ann.Rheum.Dis. 22:410.

Ross, R.F. (1973). Pathogenicity of swine mycoplasmas. Ann.N.Y. Acad.Sci. 225:347.

Rovers, M.J.C., Coovadia, H.M. (1981). Systemic Lupus Erythematosus in children : a report of 3 cases. S.Afr.Med.J. 60:711.

Runge, F. (1873). Zur Genese und Behandlung des Schreibekrampfes. Berliner Klin Wchnschr. 10:245.

Sament, S., Klugman, L.H. (1957). Dermatomyositis case report. S.Afr.Med.J. 31:430.

Saville, P.D. (1967). Symptomatic osteoporosis at the menopause. Clin.Orthop. 55:43.

Schnier, M.H., Sims, F., Zail, S. (1972). The Lesch-Nyhan Syndrome : first case description in a South African family. S.Afr.Me .J. 46:947.

Schorn, D., Welke, H., Anderson, I.F. (1975). Pseudogout - CPPD arthropathy. Case Reports. S.Afr.Med.J. 49:1266.

Schrire, V. (1958). The racial incidence of heart disease at Groote Schuur Hospital II Hypertension and valvular disease of the heart. Am.Heart.J. 56:742.

Schulz, E.J., Findlay, G.H., Scott, F.P. (1962). Skin disease in the Bantu : a survey of 4000 cases from the Transvaal and Orange Free State. S.Afr.Med.J. 36:199.

Schulze, B.V. (1973). Myalgia and arthritis in clinical medicine in Africans in South Africa. Edited by Campbell, G.D., Seedat, Y.K., Daynes, G. Churchill Livingstone, Edinburgh, London.

Schweitzer, G. (1970). Laxity of metacarpophalangeal joint of the thumb : an inter racial study. S.Afr.Med.J. 44:246.

Schweitzer, G., Jones, B., Timme, A. (1971). Upington Disease : a familial dyschondroplasia. S.Afr.Med.J. 45:994.

Seedat, Y.K., Reddy, J. (1974). A study of 1000 South African nonnwhite hypertensive patients. S.Afr.Med.J. 48:816.

Seedat, Y.K., Randeree, M. (1975). Avascular necrosis of the hip joints in hypothyroidism. S.Afr.Med.J. 49:2071.

Seedat, Y.K., Pudifin, D.J. (1977). Systemic Lupus erythematosus in Black and Indian patients in Natal. S.Afr.Med.J. 51:335.

Seedat, Y.K., Seedat, M.A., Veale, M.T. (1980). The prevalence of hypertension in urban Whites. S.Afr.Med.J. 57:1025.

Seedat, Y.K., Hackland, D.B.T., Mpontshane, J. (1981). The prevalence of hypertension in rural Zulus. S.Afr.Med.J. 60:7.

Seftel, H.C., Malkin, C., Schmaman, A., Abrahams, C., Lynch, S.R., Carlton, R.W., Bothwell, T.H. (1966). Osteoporosis, scurvy and siderosis in Johannesburg Bantu. Brit.Med.J. 1:642.

Seftel, H.C., Johnson, S., Muller, E.A. (1980). Distribution and biosocial correlations of blood pressure levels in Johannesburg Blacks. S.Afr.Med.J. 57:313.

Sharp, G.C., Irvin, W.S., May, C.M., Holman, H.R., McDuffie, F.C., Hess, E.V., Schmid, F.R., (1976). Association of antibodies to ribonucleoprotein and Sm antigens with mixed connective tissue disease, systemic lupus erythematosus and other rheumatic diseases. N.Engl.J.Med. 295:1149.

Sheldon, P.J.H. (1972). A retrospective survey of 102 cases of shoulder pain. Rheumatol.Phys.Med. 11:422-427.

Shepherd-Wilson, W., Gelfand, M. (1962). Gout in the African. Centr.Afr.J.Med. 8:181.

Shichikawa, K. (1968). Prevalence of the rheumatic diseases in Japan in Population studies of the rheumatic diseases. Edited by Bennett, P.H. and Wood, P.H.N. Excerpta Medica Foundation, Amsterdam. p.55.

Shine, I.B. (1965). Incidence of hallux valgus in a partially shoewearing community. Brit.Med.J. 1:1648.

Short, C.J. (1974). The antiquity of rheumatoid arthritis. Arthritis Rheum. 17:193.

Silbert, M.V. (1970). The Cape morbidity survey and its significance in training for general practice. S.Afr.Med.J. 44:Suppl.3-28.

Sim-Fook, L., Hodgson, A.R. (1958). A comparison of foot forms among the non-shoe and shoewearing Chinese population. J.Bone Jnt. Surg. 40A:1058.

Singer, J.M. (1974). Standardisation of the latex test for rheumatoid arthritis serology. Bulletin on Rheumatic Diseases. 24:762.

Singh, M., Riggs, B.L., Beabout, J.W., Jowsey, J. (1973). Femoral Trabecular pattern index for evaluation of spinal osteoporosis : a detailed dethodological description. Mayo Clin.Proc. 48:184.

Siri, W.E. (1961). Techniques for measuring body composition. Edited by Brozek, J. and Henschel, A. Nat.Acad.Sci. 223. 1961.

Sita, J., Sebo, M. (1968). Rheumatoid arthritis and ankylosing spondylitis in Czechoslovakia in Population studies of the Rheumatic Diseases edited by Bennett, P.H. and Wood, P.H.N. Excerpta Medica Foundation, Amsterdam.

Smith, D.M., Johnston, C.C., Yu, P.L. (1972). In vivo measurement of Bone mass. J.Amer.Med.Assoc. 219:325.

Smith, J.W., Sandford, J.P. (1967). Viral arthritis. Ann.Int. Med. 67:651.

Sokoloff, L. (1980). The pathology of osteoarthritis and the role of ageing in the aetiopathogenesis of osteoarthrosis. Edited by G.Nuki. Pitman Medical. p.1.

Solheim, B.G., Larsen, R.A. (1972). Family studies in systemic lupus erythematosus IV. Presence of antinuclear factors in the total population of relatives and spouses and the correlation to rheumatic disease. Acta Med.Scand (Suppl) 543:43.

Solomon, L. (1968). Osteoporosis and fracture of the femoral neck in South African Bantu. J.Bone Jnt.Surg. 50B:2.

Solomon, L. (1973). Drug induced arthropathy and necrosis of the femoral head. J.Bone Jnt.Surg. 55B:246.

Solomon, L., Beighton, P., Lawremce J.S. (1975). Rheumatic disorders in the South African Negro Part II osteoarthrosis. S.Afr.Med.J. 49:1737.

Solomon, L., Beighton, P. (1975). Rheumatic disorders in the South African Negro III Idiopathic necrosis of the femoral head. S.Afr.Med.J. 49:1825.

Solomon, L., Beighton, P., Valkenburg, H.A., Robin, G., Soskolne, C.L., (1975). Rheumatic disorders in the South African Negro. 1. Rheumatoid arthritis and ankylosing spondylitis. S.Afr.Med.J. 49:1292.

Solomon, L., Robin, G., Valkenburg, H.A. (1975). Rheumatoid arthritis in an urban South African Negro population. Ann.Rheum.Dis. 34:128.

Solomon, S. (1979). Bone density in ageing Caucasian and African populations. Lancet 2: 1326.

Spaar, G.S. (1964). Inheritance of flexed fingers. J.Hered. 37:189.

Sparks, L.T., Dall, G. (1982). Idiopathic chondrolyso of the hip joint in adolescents : case reports. S.Afr.Med.J. 61:883.

Spring, M., Fleck, H., Cohen, B.D. (1970). Dupuytrens Contracture : warning of diabetes. New York State J.Med. 70:1037.

Stastny, P. (1978). Association of the B cell allo-antigen DRW4 with rheumatoid arthritis. N.Engl.J.Med. 298:869.

Stewart, S.M., Alexander, W.R.M., Duthie, J.J.R. (1969). Isolation of diphtheroid bacilli from synovial membrane and fluid in rheumatoid arthritis. Ann.Rheum.Dis. 28:477.

Swannell, A.J., Dixon, A. St.J. (1969). Extra articular calcification mimicing acute arthritis. Ann. Rheum.Dis. 28.678.

Taylor-Robinson, D., Taylor, G. (1976). Do mycoplasmas cause rheumatic disease? Infection and Immunology of the rheumatic disease. Edited by Dumonde, D.C. Oxford. Blackwell scientific publications. p.177.

Temtamy, S.A., McCusick, V.A. (1978). The genetics of Hand Malformations. Birth defects original article series Vol.14. p.441. New York. A.R. Liss Inc.

Thandroyen, F.T., Matisson, R.E., Weir, E.K. (1978). Severe aortic incompetence caused by Systemic Lupus Erythematosus. S.Afr.Med.J. 54:166.

Thomas, A.F., Solomon, L., Rabson, A., (1975). Polyarthritis associated with Yersinia enterocolition infection. S.Afr.Med.J. 49.18.

Thomson, A.J.G., Bhettay, E., Steven, P. (1976). Ocular manifestations of juvenile rheumatoid arthritis. Paper presented at the 5th South African Rheumatism and Arthritis Association Congress, Cape Town May 10-14th.

Thompson, G.R., Ming Ting, Y., Riggs, G.A., Fenn, M.E., Denning, R.M. (1968). Calcific tendonitis and soft tissue calcification resembling gout. J.Am.Med.Assoc. 203:464.

Thompson, M. (1961). Shoulder-hand syndrome. Proc.R.Soc.Med. 54:679.

Truswell, A.S.)1958). Osteopetrosis with syndactyly a morphological variant of Albers-Schonberg disease. J.Bone.Jnt.Surg. 40B:208.

Tzonchev, V.T., Pilossof, T., Kanev, K. (1968). Prevalence of inflammatory arthritis in Bulgaria in Population studies of the Rheumatic Diseases. Edited by Bennett, P.H. and Wood, P.H.N. Excerpta Medica Foundation. Amsterdam/New York. p.60.

Urist, M.R. (1964). Accelerated ageing and premature death of bone cells in osteoporosis in Dynamic studies of Metabolic Bone Disease. Edited by Pearson, O.H., Joplin, G.F. p.127. Blackwell Scientific Publications. Oxford.

Upton, A.R.M., McComas, A.J., (1973). The double crush in nerve entrapment syndromes. Lancet 2:359.

Valkenburg, H.A. (1963). Human erythrocyte agglutination test (HEAT) in the Epidemiology of chronic rheumatism Vol.1. Edited by Kellgren, J.H., Jeffrey, M.R., and Ball. J. Blackwell Scientific Publications. Oxford 1963. p.330.

Vaughan, J.H., Catalano, M.A., Jensen, F.C., Carson, D.A. (1978). Antinuclear antibodies in rheumatoid arthritis with special reference to those specific for B Lymphocytes infected with Ebstein -Barr virus in Immunopathogenesis of rheumatoid arthritis. Edited by Panayi, G.S., and Johnson, P.M. Reedbooks Ltd., Surrey p.72.

Verzar, F. (1957). Connective Tissue - a symposium C.I.O.M.S. Edited by Tunbridge, R.E. Blackwell Oxford. page 208.

Viljanto, J.A. (1973). Dupuytrens Contracture - a Review. Sem.Arthritis Rheum. 3:155.

Virchow, R. (1858). Die cellular pathologie in ihrer Begründing auf physiologische und pathologisches Gewebelehre. A.Hirshwald. Berlin.

Wahner, H., Riggs, B.L., Beabout, J.W., (1977). Diagnosis of osteoporosis : usefulness of photon absorptiometry at the radius. J.Nuclear Med. 18:432.

Walford, R.L. (1962). Autoimmunity and ageing. J.Gerontol. 17:281.

Walker, A.R.P., Walker, B.F., Richardson, B.D., Christ, H.H. (1970). Cortical thickness of bone in underpriveleged populations. Am.J. Clin.Nutr. 23:244.

Walker, A.R.P., Walker, B.F., Richardson, B.D. (1971). Metacarpal bone dimension in young and aged South African Bantu consuming a diet low in Calcium. Postgrad.Med.J. 47:320.

Weinstein, A., Bordwell, B., Rothfield, N. (1978). Anti-native DNA antibodies and serum C3 levels. Candidates for the ARA preliminary criteria for the classification of systemic lupus erythematosus. (Anstr.). Arthritis Rheum. 21:602.

Weiss, G., (1964). Sarkoïedose : 'n geval bespreking en oorsig, vandie jongste literatuur. Geneeskunde 6:296.

Weissenberger, R., Robertson, A., Holland, S., Hall, W. (1977). The incidence of gonorrhoea in urban Rhodesian Black women. S.Afr.Med.J. 52:119.

Welch, J.P., Temtamy, S.A. (1966). Hereditary contractures of the fingers (camptodactyly). J.Med.Genet. 3:104.

Whittingham, S., McKay, I.R. (1969). Laboratory methods for the diagnosis of auto-immune disease. Med.J.Aust. 1:1200.

Whittingham, S., Irwin, J., MacKay, I.R., Marsh, Cowling, D.C. (1969). Auto antibodies in healthy subjects. Aust.Ann.Med. 18:130.

Wielinga, W.J. (1961). Dupuytrens contracture. Arch.Clin.Neerl. 13:319.

Wood, J.W., Kato, H., Johnson, K.G., Uda, Y., Russell, W.J., Duff, I.F. (1967). Rheumatoid arthritis in Hiroshima and Nagasaki, Japan : prevalence incidence and clinical characteristics. Arth.Rheum. 10:21.

Wood, M.G., Beerman, H. (1960). Necrobiosis lipoidica granuloma annulare and rheumatoid nodule. J.Investig.Dermatol. 34:139.

Wood, P.H.N. (1969). Epidemiology of Rheumatic Disorders : Problems in classification. Proc.Roy.Soc.Med. 63:189.

Wood, P.H.N. (1977). The scope of the problem. Ulster Med.J. 47.Suppl.1.

Wood, P.H.N. (1977). The challenge of arthritis and rheumatism. The British League against Rheumatism.

Woods, D.L. De v. Heese, H., Davey, D.A., Van Schalkwyk, D.J. (1978). Stature and weight of Coloured primigravidas in Cape Town. S.Afr.Med.J. 54:776.

Woolsey, I.D., (1952). Prevalence of arthritis and rheumatism in the United States. Publ.Health Rep. Washington 67:505.

Zingale, S.B., Minzer, L., Rosenberg, B., Lee, S.L. (1963). Drug induced lupus syndrome : clinical and laboratory syndrome similar to systemic lupus erythematosus following anti-tuberculous therapy. Arch.Int.Med. 112:63.